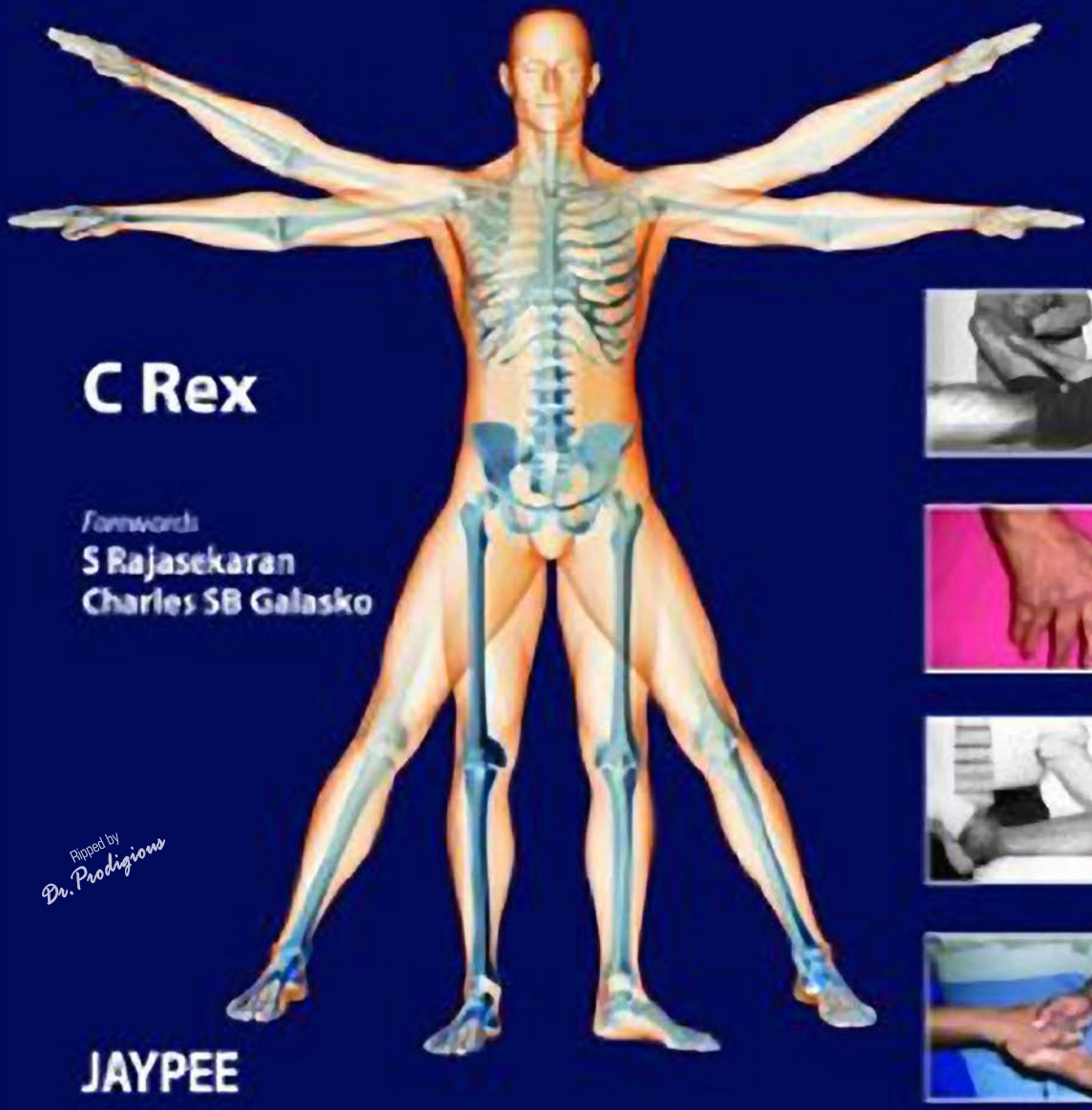
Clinical Assessment & Examination in ORTHOPEDICS

2nd Edition



C Rex

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SECOND EDITION

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Clinical Assessment and Examination in Orthopedics

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Dedicated to

The wisdom of my teachers,
The gratitude of my patients,
The love of my parents and
my family who have immeasurably enriched my life

Foreword to the Second Edition

The basis of successful management of any orthopedic patient depends on a firm diagnosis, assessment of his disability and understanding of his needs and expectations. Although modern advances in diagnostic and imaging modalities have opened up new and exciting avenues for evaluating a disease, these can never replace good history taking and a sound clinical examination. Investigations like MRI are so sensitive that many normal changes are projected as pathology leading to over-diagnosis and many unnecessary treatment. Further, patients with similar imaging results have different disability and symptoms. It is always wise to fully understand the patient and evaluate the disease clinically before any plans for treatment is made.

I am glad to note that each chapter has been written in a systematic pattern starting from good history taking and proceeding to a stepwise clinical examination following the wise principle of 'look, feel, move and document'. Each chapter also has clinical tests specific to the region and pathology well explained and illustrated. The theoretical basis of each of these tests is also clearly explained and this will be very useful to young doctors in training.

I have known the author Dr Rex personally for many years and being the good clinician that he is, it is no wonder that he has brought out this book which would not only be useful to training doctors but also be a ready-reference to all practicing orthopedic surgeons.

S Rajasekaran
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Foreword to the First Edition

The basis of orthopedic surgery is the clinical examination, including history of the patient followed by the relevant investigations. Unless the clinician is able to take a proper history and conduct the clinical examination in a systematic fashion, he/she will neither be able to make the correct diagnosis nor be aware of the relevant necessary investigations required to reach the correct diagnosis and discuss the optimum management with the patient. This book is aimed at undergraduate students and orthopedic trainees and serves its purpose in detailing the techniques of examination of the different regions. The basis of the examination of each region is based on the history, followed by Alan Apley's classical method of examination—look, feel, move. The author and the editors of the individual chapters, all of whom are experienced surgeons in their specific field, have then described specific tests and conditions. Orthopedics is a visual subject with inspection playing an important role in clinical examination. It is for this reason that the book has been well illustrated, such that the student will gain much more from the text.

We live in an era of increasingly sophisticated investigations and the development of molecular biology, genetics, tissue engineering, etc. all of which will have a major impact in the delivery of orthopedic services but if the patient is to obtain the maximum benefit from these developments which are likely to revolutionize the practice of orthopedic surgery, the orthopedic surgeon must still be able to examine the patient and make a diagnosis. Unless the surgeon can reach a reliable differential diagnosis, the relevant investigations are not likely to be undertaken and the correct diagnosis is not likely to be made. Unless the correct diagnosis is made, the advances in orthopedic surgery will be meaningless. The basis of diagnosis will continue to be the clinical examination as laid down in each chapter of this book.

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Past Vice President, Royal College of Surgeons of England
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Sir Arthur Sims, Commonwealth Professor

Preface to the Second Edition

Overzealous response from orthopedic trainees and recent advances has made me to write this second edition. Though examination techniques are same, the recent thinking and diagnosis of various new conditions because of explosion of knowledge and array of investigation has thrown light to many new facts compelling a clinician to diagnose by simple examination. As medicine is evolving and advancing everyday our understanding on clinical assessment and examination has also improved a lot.

The book is aimed at orthopedic trainee and junior consultants to sharpen their clinical skills and to follow a methodical approach in examination. I have taken every effort to update this second edition with more illustrations on demonstration techniques, clinical pictures with obvious diagnosis and additions of some of the advancement made in subspecialties like shoulder and wrist.

The book must be simple and easy for understanding so that it enables a trainee to rapidly acquire knowledge in history taking and examination in a systematic way. Repeated practice will bring perfection and, when one masters the technique it takes no time to arrive at a right diagnosis. I have put together all the essential parts of clinical assessment in a nutshell to diagnose common orthopedic conditions. In history taking, importance is given for patient's disability, patient's perception of nature of illness and patient's expectation in order to device a treatment plan. The sequence of examination has been described in a more practical way applying the conventional system of look, feel and move, and when patient stands, sits and lies down.

The doctor can win the patient's confidence by the way he/she communicates to the patient, handling the patient with utmost care and by a smooth clinical examination. Well dressed doctor with a compassionate approach and an authoritative talk to the patient is essential to win the patient's confidence. A short note on clinical findings has been described on common disease conditions at the end of each chapter.

Every attempt has been made to give self-explanatory illustrations on examination techniques. Each chapter has been edited by eminent experts in their respective field to keep pace with the standard and recent advances in their field, so that the reader gets the maximum benefit. I welcome suggestions to bring out an even better next edition by filling the lacunae. I hope this book will fulfill the requirement of an orthopedic trainee.

C Rex

Preface to the First Edition

As medicine is evolving and advancing everyday, our understanding on clinical assessment and examination has also improved a lot. This book is aimed at orthopedic trainee and junior consultants to sharpen their clinical skills and to follow a methodical approach in examination.

I have taken every effort to make this book simple and easy for understanding so that it enables a trainee to rapidly acquire knowledge in history taking and examination in a systematic way. Repeated practice will bring perfection and, when one masters the technique it takes no time to arrive at a right diagnosis. I have put together all the essential parts of clinical assessment in a nutshell to diagnose common orthopedic conditions. In history taking, importance is given for patient's disability, patient's perception of nature of illness and patient's expectation in order to device a treatment plan. The sequence of examination has been described in a more practical way applying the conventional system of look, feel and move when patient stands, sits and lies down. The doctor can win the patient's confidence by the way he/she communicates to the patient, handling the patient with utmost care and by a smooth clinical examination. A short note on clinical findings has been described on common disease conditions at the end of each chapter. Every attempt has been made to give self-explanatory illustrations on examination techniques. Each chapter has been edited by eminent experts in their respective field to keep pace with the standard and recent advances in their field, so that the reader gets the maximum benefit. I welcome suggestions to bring out an even better next edition by filling the lacunae. I hope this book will fulfill the requirement of an orthopedic trainee.

C Rex

Acknowledgments

I would like to acknowledge my teachers, fellow colleagues, my team of Orthopedic Junior Consultants and trainees for the guidance and help received in the preparation of this manuscript.

I immensely thank Professor S Rajasekaran, Department of Orthopedics, Ganga Hospital, Coimbatore, Tamil Nadu, India for readily accepting to write the foreword for the second edition and spending his valuable time in spite of his busy schedule. He has been my guiding light and mentor. I vow him a lot for what I am now.

I would like to thank Mr Reginald T, our Senior Physiotherapist for compiling and timely preparation of the manuscript.

This project could not have been successfully completed within the prudent and dutiful efforts of the many individuals whom I acknowledged in the previous edition and the present edition here.

Contents

1 CHAPTER History Taking and Clinical Examination

The art of history taking and clinical examination in a systematic fashion should be learnt to arrive at a right diagnosis and management. This is learned over a period of time and by repeated practice.

Looking at the patient as he/she walks into the room, gives an overall picture and sometimes the personality of the patient. Introduce yourself to the patient; ask the name of the patient, accompanying attendees and their relationship. Some patients may feel more shy and embarrassed to talk of their problems in front of others. Patient's inhibition to discuss in front of others must be appreciated, and, if necessary, more private and confidential discussion should be held.

The doctor must be very alert and listen to each and every word, the patient says. It is a good practice to document the history as the patient relates it.

The doctor must have lot of patience and perseverance with difficult patients in eliciting history. Conversation should be guided, avoiding any leading questions. The questions should be worded in simple language and patient-understanding of the questions should be ensured.

Always explain to the patient what you are going to do and why you are doing it, in all stages of examination to alleviate anxiety. Eye to eye contact, getting attachment to patient's version of the cause and extracting the major problem for which the patient has come today is important. Patient must be examined gently, without eliciting pain and the environment made comfortable. The doctor should be confident and impressive to the patient, and the patient must feel he/she is in the right place, in safe hands and getting good care.

HISTORY TAKING

Present Complaint

Ask the patient what is his main problem and what made him to come and see you, the duration of each salient complaint must be charted in chronological order.

History of Present Complaint

The full details of the presenting complaint from the time it started must be asked for, the progression of the symptoms, severity and associated symptoms must be recorded. Quantify the disability due to the problem in terms of day-to-day activities, job-related or hobbies and ask the patient what activities he/she cannot do? Questions about the abnormal system or any symptoms of possible diagnosis must be asked for.

Previous History

This should include any similar problems in the past, illness like diabetes, hypertension, rheumatism, asthma, allergy, tuberculosis, chest and heart problems, and dyspepsia or peptic ulcer disease (as most of the patients need anti-inflammatory tablets).

Treatment History

Enquire about all the treatments the patient has had including medicines like nonsteroidal anti-inflammatory drugs, steroids, physiotherapy, plasters, orthosis, intra-articular steroid injections, etc. and find out the effect of each treatment. Ask for any allergy to medicines. History of previous surgeries are important.

Family History

Enquire about the general family health, occurrence of any familial or hereditary diseases, and support from the family in terms of psychological and financial aspects.

Social and Occupational History

Marital status, type of place where he/she lives, presence of stairs at home and toilet facilities (Indian or Western toilet—this is important in patients with hip or knee pathology) must be recorded. History of consanguineous marriage is important in the presence of congenital anomalies in their offspring. Exact nature of occupation—sedentary or heavy manual work, the hobbies and the patient's leisure activities must be noted.

Personal History

Alcohol consumption, smoking habits, dietary habits and sexual life must be recorded. Always quantify the amount of smoking and alcohol intake.

Patient's Expectation

The patient's expectation is an important factor in the treatment plan. Expectation of each patient is different and sometimes may not be realistic. Some patients might seek an advice just for reassurance rather than for treatment. This must be identified and treated accordingly.

History of Pain

Pain is what the patient feels and *tenderness* is what the doctor elicits. As pain is an important symptom which gives a clue to the diagnosis, it must be evaluated in detail.

a. *Site of pain—localized or diffuse*: Ask the patient to denote the maximum point of pain and also the extent of pain.
b. *Time and mode of onset*: It is good to know what triggered the pain at the time of onset and find out what patient was doing at that time. The pain might have begun suddenly or insidiously.
c. *Severity of pain*: This can be assessed in patient's own words as mild, moderate and severe. Find out whether the patient is able to carry out the daily routine and can perform the job, this again indicates the severity. Does the pain wake-up or keep the patient awake at night or does it force the patient to lie still, this also indicates the severity.
d. *Nature of pain*: It is good to qualify the pain as aching, stabbing, burning, throbbing, constricting or gripping pain, or pricking pain.
e. *Progression of pain*: Has the pain gone worse, remained same or decreased in time? Is the pain constant or present on and off?
f. *Radiation*: Find out the direction and exact site of radiation from the site of origin.
g. *Aggravating and relieving factors*: Ask this question directly to the patient and also document what happens to the pain on joint movements, on walking, standing, body posture and exercises. Is this pain related to any food intake (e.g. gout)? The relief of pain with analgesics, fomentation and other means should be noted.
h. *Patient's opinion on cause of pain*: This may throw some light on the cause and also some insight into the patient's problem.

History of Swelling

a. *First notice*: When did the patient notice the swelling or lump (it may not be the time when it first appeared). Patient might have noticed due to pain or might have noticed at the time of bath, or someone might have pointed it out.

b. *Symptoms associated with lump*: Pain, pressure symptoms—neurological, vascular or affecting movements of adjacent joints.
c. *Progression of the lump*: Getting bigger or smaller, or disappearing and reappearing in different positions, at different times, etc.
d. *Any other swelling*
e. *Patient's opinion on cause of swelling.*

Similar sequence is followed in the history of an ulcer.

The history most of the time gives the most probable diagnosis. An experienced clinician modifies the examination by specifically looking for signs that will confirm or refute the provisional diagnosis. If clinical signs do not favor the diagnosis then he returns to the normal routine. But all students must strictly follow the pattern of examination. Also be aware, common problems are common. Do not think of rare diagnosis as we may be rarely right and keep things simple and easy.

Clinical Examination

General Examination

The general examination analyzes the patient as a whole. General build, mental state, presence of anemia, jaundice, cyanosis, clubbing, skin and nail changes (pitting in Psoriasis), pedal edema, fever, multiple bone or joint deformities and any generalized manifestation of a disease must be recorded.

Local Examination

Examining the area of symptomatology and examination of appropriate system causing the symptoms add more information of the disease and the possible diagnosis. This follows the foolproof systematic approach of:

- Inspection (Look)
- Palpation (Feel)
- Movements—Active and passive movements. Joint range of movements are measured with goniometer
- Measurements
- Neurovascular status

Local examination of each part of locomotor system is discussed in detail in subsequent chapters.

EXAMINATION OF SWELLING

- Site
- Size
- Color **(Figs 1.1 and 1.2)**.
- Temperature
- Tenderness
- *Shape:* Hemispherical, oblong, kidney-shaped, pear-shaped, etc.
- *Surface:* Smooth, irregular, bosselated or lobular
- *Edge:* Well-defined, indistinct
- *Consistency:* Soft (like an ear lobe), firm (like tip of nostril), hard bony hard, variegated–different consistency in different parts of swelling

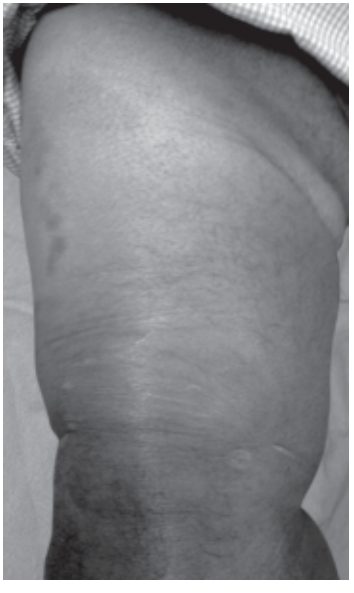


Fig. 1.1: Spreading cellulitis of thigh marked by redness, swelling and warmth of skin and subcutaneous tissue

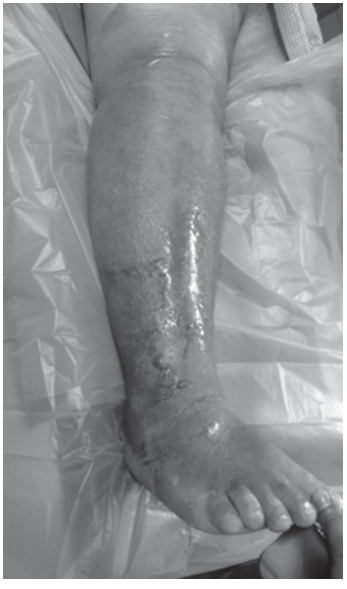


Fig. 1.2: Necrotizing fascitis showing blackening and blistering of skin due to gangrene with redness above *(For color version, see Plate 1)*

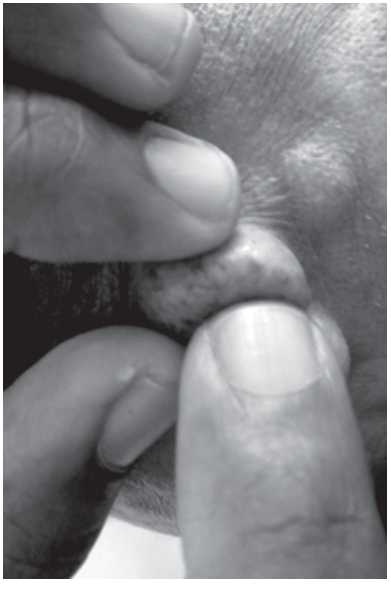


Fig. 1.3: Paget's test: Forehead swelling of 2 cm tested for fluctuation by fixing the swelling with examiner's index finger and thumb and with the other hand index finger the center of the swelling was pressed to feel the fluid displacement *(For color version, see Plate 1)*

- *Fluctuation:* In cystic swelling small tense swelling is tested for fluctuation by fixing the swelling's outer margin with one hand and with one finger press on the middle of the swelling to elicit fluctuation—Paget's test **(Fig. 1.3)**. Large cystic swelling should be checked for cross-fluctuation in two planes (right angle to each other) to differentiate from pseudo- fluctuation in soft swelling like lipoma.
- *Reducibility*
- *Pulsatility:* True expansile pulsations are from aneurysms and vascular tumors. Transmitted pulsations can be felt on swelling over major arteries.
- *Transillumination:* Light will pass through clear fluid. Using a pen torch in a darkroom one can demonstrate a flare in clear fluid-filled sac.
- *Plane of the swelling:* Relationship to surrounding structures—Can skin be pinched separately? Swelling deep to muscle becomes less prominent on muscle contraction and difficult to move. Swelling superficial to muscle remains same in size and have free mobility.
- *Fixity:* To skin or bone.
- Regional lymph nodes
- *State of arteries, nerves, bones and joints.*

EXAMINATION OF ULCER

- *Site*
- *Size, shape*
- *Color*
- *Warmth*
- *Tenderness*
- *Base or floor:* It is the surface of the ulcer. It can be of healthy red granulation tissue or gray dead tissue or exposed muscles/tendons
- *Edge* Sloping edge—In a healing ulcer
  Punched out edge—In trophic ulcer (syphilis, neuropathy, spina bifida)
  Undermined edge—In tuberculosis
  Rolled out edge—In basal cell carcinoma

Everted edge—In squamous cell carcinoma

- *Depth:* The fixity to deep structures is indicated by the mobility of the ulcer
- *Discharge:* Serous or serosanguinous or purulent
- Regional lymph node
- *State of arteries, nerves, bone and joints.*

EXAMINATION OF BONE AND SOFT TISSUE TUMORS

Bone sarcomas are common in children and adolescents while secondaries and myelomas are common in elderly population.

Presenting History

1. *Pain*—Onset, duration, nature, aggravating and relieving factors.
2. *Swelling*—Onset, duration, progress, change in size and other swellings.
3. *Pathological fracture*—Trivial injury causes fracture.
4. *Distant site problems*—Symptoms from metastasis: lung symptoms, symptoms of hypercalcemia or neurological deficit from local spread.

Past History

Any significant medical illness, previous malignancies treated like carcinoma prostate, lung, thyroid, kidney, etc., radioactive isotope treatment or irradiation.

Family History

Hereditary disease like autosomal dominant von Recklinghausen's disease, diaphyseal aclasis, multiple lipomata, etc.

Personal History

Smoking, alcohol, mental status and social background.

Occupational History

Exposure to radioactive materials, chemicals, etc.

General Examination

General build, anemia, jaundice, cyanosis, clubbing, generalized lymphadenopathy.

Systemic Examination

Examination of the lungs, abdomen, pelvis and nervous system, if necessary.

Local Examination (Fig. 1.4)

Examination of the swelling is done as described before, special points to be noted in tumors are:

1. Aggressiveness—A tense rapidly growing tumor with shiny skin, engorged veins and variable consistency is typical of aggressive tumor.
2. Skip lesions—Look for satellite lesions in the same extremity.
3. Pressure effects—Neurovascular impairment or limitation of range of movements.
4. Regional lymph node involvement.
5. Auscultation—Listen for a bruit in suspected vascular swellings, telangiectatic osteogenic sarcoma, vascular secondaries, etc.

Origin of tumors in various parts of bone is illustrated in **Figure 1.5**.

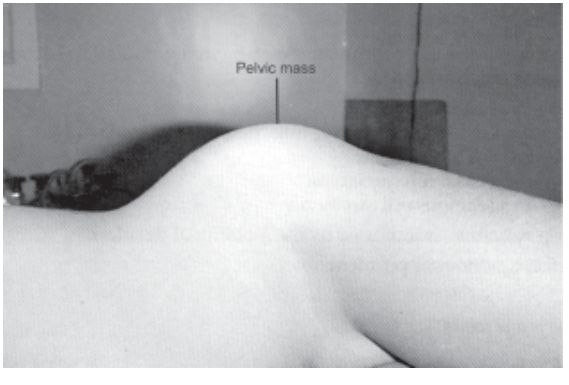


Fig. 1.4: Ewing's sarcoma from pelvis

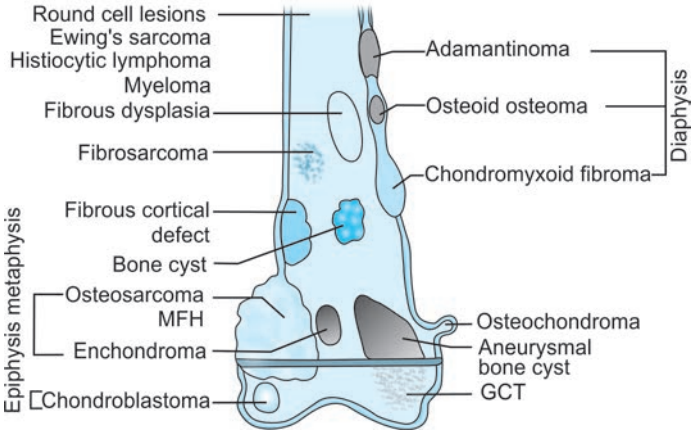


Fig. 1.5: Origin of tumors in various parts of bone

EXAMINATION OF BONE AND JOINT INFECTION

Acute Osteomyelitis/Joint Sepsis

This is more common in children but can occur in adults who are immunosuppressed or drug addicts. Child who is not well, irritable, having high fever, suddenly not moving the limb (pseudo-paralysis) with septic foci in the body should arise high suspicion of acute osteomyelitis or joint sepsis. Joint sepsis is more common in the hip, which presents with inability to move the limb, flexion attitude of the limb, gross restriction of movements with pain and spasm. Patient or parents may attribute it to a fall.

Acute osteomyelitis presents with sudden onset of pain, swelling, inflammation and loss of function and should be diagnosed clinically.

Both osteomyelitis and joint sepsis should be treated aggressively by investigating full blood count, ESR and blood culture with antibiotics, and if necessary, emergency surgical drainage. Ultrasound scan may be of good help to establish the diagnosis.

Chronic Osteomyelitis (Figs 1.6A and B)

Patient can present with pain, discharging sinus, difficulty in using the limb or weightbearing in the lower limb, pathological fracture or exuberant growth from ulcer (Marjolin's ulcer).

In the history onset of first episode, the progress, the treatment taken including various antibiotics and surgical procedures must be taken elaborately. Osteomyelitis starting in childhood will naturally affect the growth of the bone and can cause soft-tissue tightness. History of any immunosuppressive disease or drugs (steroids/chemotherapy), smoking, alcohol, diabetes, sickle cell disease and tuberculosis must be asked for. Enquire about constitutional symptoms, weight loss, etc.

General Examination

General build, anemia, jaundice, cyanosis, generalized lymphadenopathy, septic focus in the body.

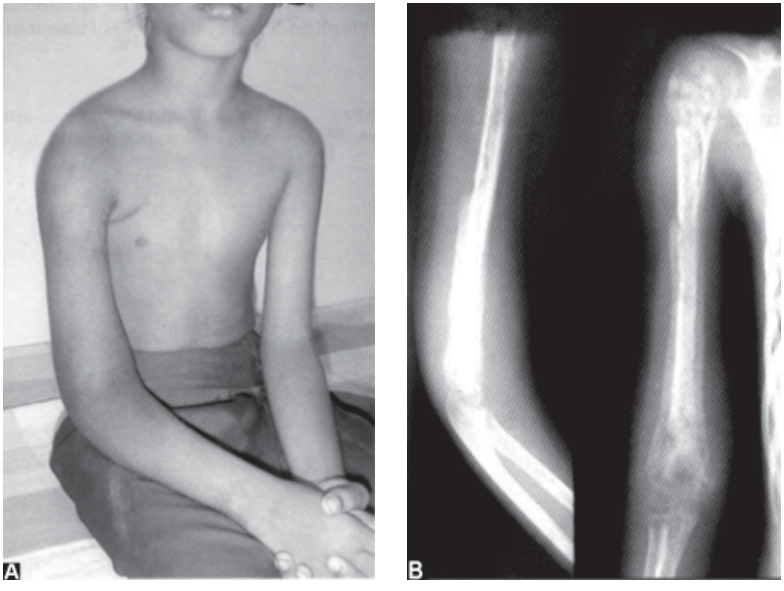
Local Examination

Look

Look for gait (in lower limb involvement), deformity, leg length discrepancy, skin and soft tissue status—ulcer or sinus, puckered scar or wound healed by secondary intention.

Feel

Feel for warmth, tenderness in soft tissue and bones, bony thickening, soft tissue indurations,



Figs 1.6A and B: Diaphyseal sequestrum in chronic osteomyelitis of humerus

percussion of the bone causes deep severe pain, abnormal mobility in pathological fracture or infected non-union, adjacent joints for stiffness and deformity, distal neurovascularity, regional lymph nodes and other focus of infection.

Move

Test active movements first and then passive movements. Do not attempt movement in an acute infection (very painful). Look for stiffness, abnormal mobility, subluxation or dislocation in chronic infection.

2 CHAPTER Examination of Injured Patient

INTRODUCTION

Trauma is the leading cause of death in the first four decades of life and the third most common in all ages. In addition to the deaths caused many more people are temporarily or permanently disabled as a result of accidents.

Clearly the best way of approaching the management of trauma is prevention or the minimization of its effects (e.g. compulsory use of motorcycle helmets). These areas lie with governmental bodies and equipment manufacturers. The physician's main role is to identify and treat any injuries sustained, while preventing secondary injuries occurring.

These latter two areas have been addressed with the inception of the Advanced Trauma Life Support (ATLS®) course in 1980 and its subsequent revisions. The traditional approach to treatment of patients is to take a full history with examination and perform investigations prior to treatment, as taught at medical schools. However, the ATLS® approach seeks to identify and simultaneously treat immediate life-threatening conditions in a predictable order. The aim of this section is to describe the assessment of a patient with musculoskeletal trauma according to the ATLS® framework of trauma management. A formal review of the ATLS® course is not appropriate here and the reader is recommended to attend a locally organized course.

INITIAL MANAGEMENT OF THE TRAUMA PATIENT

Managing a trauma patient follows a logical sequence aimed at simultaneously identifying, treating then re-evaluating life-threatening injuries, which is followed by a detailed survey for all injuries sustained **(Table 2.1)**.

Airway with Cervical Spine Control

On first contact with the patient, the airway is checked at the same time as protecting the cervical spine. The patient is asked if they are all right and an appropriate response will quickly confirm that there is an adequate airway with ventilation and cerebral perfusion. The cervical spine is protected with a rigid collar, head blocks and tapes. A spine board may be used for transfer purposes, but it should be removed as quickly as possible to prevent pressure sores developing. Initial airway management involves clearing the upper airway of obstructions (e.g. saliva, blood, dentures or teeth) and improving its patency, by simple airway maneuvers (e.g. jaw thrust or chin lift supplemented with an oral or nasopharyngeal

Table 2.1: ATLS® approach to managing the trauma patient

Primary survey

1. Airway with cervical spine control
2. Breathing
3. Circulation with hemorrhage control
4. Disability
5. Exposure
6. Re-evaluation
7. Adjuncts to the primary survey

Secondary survey

1. History
2. Examination
3. Adjuncts to the secondary survey

airway). Supplemental oxygen (15 liters per minute via a mask with reservoir bag) is given. A definitive airway is one where a cuffed endotracheal tube has to be inserted, which may be inserted via the oral or nasal route. A surgical airway may be created in the emergency situation by cricothyroidotomy. The decision about which method of airway management is best, lies out with this discussion.

Breathing

Breathing problems follow on logically from management of the airway. The first indication of a musculoskeletal injury may be compromise to breathing due to a cervical spine injury, especially if this is above the supply to the diaphragm (C3,4,5). Treatment of such lesions in the ATLS® approach commences before breathing difficulties may have been identified, as the cervical spine has already been immobilized to prevent further injury as a part of the airway management. It should be noted that clearance of the cervical spine of injury might occur a long time after admission, even with normal cervical spine radiographs. Other causes of breathing difficulties can broadly be grouped into those due to chest injuries and those occurring outside the chest. Immediately life-threatening chest injuries include major airway obstruction, tension pneumothorax, flail chest, open pneumothorax and massive hemothorax. Other injuries lying outside the chest that can affect breathing are mainly of neurological origin and include head injuries or alcohol/drug intoxication. Routine examination should identify the majority of these injuries prior to radiographs being taken. Breathing difficulties are treated by ventilatory support as required along with specific treatment for the underlying injury, the majority of which will be by conservative means.

Other chest injuries which can be life-threatening but are often only picked up on the secondary survey are: simple pneumothorax, hemothorax, pulmonary contusions, lower airway injuries, multiple rib fractures, cardiac or major vessel injury and diaphragmatic injury. Identification of these requires examination along with additional tests, both of which are often based on a high index of suspicion and are described in detail at the end of this chapter.

Circulation with Hemorrhage Control

Further evidence of a spinal injury may also be found on assessment of the circulation, where neurogenic shock may be present. Neurogenic shock classically presents with a low blood pressure, bounding pulses, a warm periphery and sometimes bradycardia. The findings are due to peripheral vasodilatation due to loss of sympathetic tone, and should not be confused with "spinal shock" which refers to the temporary total loss of function of the spinal cord. Care must be taken in solely attributing a low blood pressure to a spinal injury, as the most common cause of hypotension following trauma is hypovolemia. Hypovolemia must be treated appropriately before attending to neurogenic shock. Hypovolemia may be caused by other musculoskeletal injuries such as major pelvic disruption (especially open book and vertical shear fractures), major vascular hemorrhage (arterial or venous) and crush injuries associated with myoglobin release causing hypovolemia, metabolic disturbances, disseminated intravascular coagulation (DIC) and renal failure. However, other common causes include intra-abdominal bleeding and massive hemothorax, it may also be mimicked by tension pneumothorax and cardiac tamponade. The other causes of shock are seen less commonly in the acute trauma situation. Evaluation again is performed by routine clinical examination, although it is often supplemented by radiographic and more complex investigations. Treatment is aimed at replacing the lost fluid (initially with crystalloid and subsequently blood) along with prevention of further blood loss (which may require splintage or direct pressure for musculoskeletal injuries or operation for visceral injuries).

Disability

Neurological compromise is often due to head injury, but may also be due to poor cerebral perfusion resulting from an inadequately treated airway, breathing or circulatory problem. Within the brain the injury may be due to either a focal or diffuse lesion. Assessment is initially performed by assessing the Glasgow coma scale, **(Table 2.2)** pupillary responses and observation of localizing signs. A more detailed neurological assessment follows later. Treatment is aimed at preventing further injury by providing adequate cerebral perfusion and oxygenation, along with identifying and treating the appropriate focal lesions by referral to the neurosurgeons.

Exposure

This precedes the comprehensive secondary survey and allows a formal full assessment of the patient. Wet, soiled or chemical coated clothes are removed and the patient is covered and warmed appropriately to ensure a normal body temperature is maintained.

Further details of the examination and treatment of all of these problems are fully addressed in the ATLS® course. In addition, variations in patient subgroups (e.g. children, the elderly and pregnancy) are also covered. Following re-evaluation of the patient adjuncts to the primary survey are performed, which include cervical spine (where appropriate), chest and pelvic radiographs. The secondary survey follows and includes the history and examination. For the purposes of this chapter these will be considered only in the light of musculoskeletal trauma.

Table 2.2: The Glasgow coma scale

Response	*Description*	*Numerical value*
Eye opening	Spontaneous response	4
	Response to speech	3
	Response to pain	2
	No response	1
Motor response	Obeying response	6
	Localized response	5
	Withdrawal	4
	Abnormal flexion	3
	Extension	2
	No response	1
Verbal response	Oriented conversation	5
	Confused conversation	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1

HISTORY

Knowledge of the mechanism of trauma is extremely important in identifying the nature and severity of specific injuries. It may also dictate the treatment plan.

The most important determinant of severity of injury is the amount of energy transferred at the time of the accident. The kinetic energy imparted ($\frac{1}{2}$ mv^2) is determined by the velocity (v) at the time of impact (e.g. simple fall *vs.* hit by a car at 70 mph) and mass (m) of the colliding bodies (e.g. handgun bullet *vs.* car). This will be modified by the direction of the applied force (e.g. glancing blow *vs.* head on impact) and the effects of any protective devices (e.g. crash helmet).

The location of the accident is of special importance as the environment can influence both general (e.g. hypothermia) and local (e.g. farmyard contamination of open injuries) factors, which may influence treatment.

Certain associations exist between some accident mechanisms and injury patterns (e.g. dashboard injury and posterior dislocation of the hip). In addition, some injury patterns are associated with each other (e.g. calcaneal fracture and lumbar spine vertebral body fracture) and where one is found the other must be excluded.

The symptoms experienced by the patient at the time of injury and immediately afterwards must be sought. Clearly, immediate pain and localized bony tenderness raises the possibility of a fracture, however, in anterior cruciate ligament injuries only a pop may be felt, but this is usually followed by immediate swelling of the knee and inability to weight bear. Associated symptoms must also be sought within all systems in the affected limb or area (e.g. median nerve symptoms following supracondylar fracture of the elbow). Finally, if there is a delay in presentation, symptoms may develop suggesting a chronic lesion (e.g. joint instability following ligament disruption).

The history is completed with a detailed past medical and surgical history, family and social history, allergies and current/previous medications. For acute injuries the time of the accident as well as the details of the last drink and meal are very important.

EXAMINATION

Examination will be considered in relation to the trauma patient and the pattern of examination is similar for injuries to all tissues **(Table 2.3)**. Although this section is artificially divided for clarity, many tests are common to more than one system or pathology (e.g. testing active range of movement assesses the bones, adjacent joints, muscles, nerve supply and vascular status). In addition, only aspects relating to trauma will be described, for a full description of the examination of a particular joint or region the reader is directed to the appropriate chapter elsewhere in this book.

Table 2.3: Pattern of examination of the musculoskeletal system

a. Inspection	
b. Palpation	
c. Movement	Active and passive
d. Special clinical tests	Ligament testing
	Neurological examination
	Rectal examination
e. Adjuncts	Doppler studies
	Compartment pressure monitoring
	Radiographs
	Ultrasound scan
	MR scan

Fractures and dislocations are probably the most commonly encountered musculoskeletal injuries. The other injuries indicated below are sometimes seen in isolation especially in penetrating trauma; however, they are more commonly seen in combination with underlying fractures or dislocations.

Fractures and Dislocations

- *Inspection:* The limb should be inspected for swelling around the fracture site and there may be an associated effusion with an underlying joint injury. Deformity of the limb may be clear although only shortening or rotation may be evident. The skin should be assessed (see below).
- *Palpation*: The limb should be palpated for tenderness without causing excessive discomfort to the patient, including pelvic springing which should be performed only once. Abnormal movement and crepitus is classically described at the site of fractures but should not be elicited. Muscle spasm may be found in some cases and is especially associated with dislocations and spinal column fractures (paraspinal muscles). Deformity may only be identified on palpation (e.g. a step in spinous processes in spinal dislocations).
- *Active then passive movements:* These movements should be attempted, but in fractures this is frequently not possible due to pain. In dislocations some movement is often possible and a block to a particular range of movement should be sought (e.g. external rotation in posterior dislocation of the shoulder).
- *Special clinical tests:* These tests include assessment of the ligaments in joint injuries. There should be routine examination of the neurovascular status of the limb (see below) to

exclude associated injuries (e.g. axillary nerve palsy after shoulder dislocation and popliteal artery injury after knee dislocation). Compartment syndrome should also be excluded (see below). Some specific injuries dictate that other areas should be assessed; these include abdominal/urological examination in pelvic fractures and complete neurological including rectal examination in spinal injuries.

- *Further tests and imaging studies:* These will be dictated by the examination findings.

Skin

- The skin overlying any injury may have been breached. Associated swelling and contamination should be assessed. In addition, it may be clear that the underlying bone has been exposed. Where open fractures are identified the severity of injury is determined not only by the size and contamination of the wound but also the degree of underlying soft tissue and bony injury. Grading of these injuries is described and that for tibial fractures is presented in **(Tables 2.4A and B)**.
- Where the skin has been breached the initial examination is really limited to inspection. No wound overlying a suspected fracture should be probed in the casualty department. It should be photographed if possible and covered with a betadine or saline soaked dressing. This dressing should not be disturbed by anyone until the patient is taken to the operating theater. Further examination should however be directed at identifying any underlying injury (e.g. tendons, nerves or vessels). Knowledge of local anatomy is imperative in determining which structures are likely to have been injured.

Muscles and Tendons

Direct injury to muscles may occur due to disturbance by fracture fragments from within or from any source of penetrating injury. Muscle belly injuries tend to be less of a problem in the long-term unless there is an additional compartment syndrome (see below) or the injury is very extensive. Tendon injuries however are more likely to occur and can result in significant disability.

Table 2.4A: Gustilo grading of open fractures of the tibia

Type I	:	An open fracture with a wound less than one centimeter long and clean.
Type II	:	An open fracture with a laceration more than one centimeter long without extensive soft tissue damage, flaps or avulsions.
Type III	:	Either an open segmental fracture, an open fracture with extensive soft tissue damage, or a traumatic amputation.

Gustilo RB, Anderson JT: Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: Retrospective and prospective analyses. *J Bone Joint Surg [Am]* 1976;58(4): 453-58.

Table 2.4B: Gustilo grading of type III (severe) open fracture

Type III A	:	Adequate soft tissue coverage of a fractured bone despite extensive soft tissue laceration or flaps, or high-energy trauma irrespective of the size of the wound.
Type III B	:	Extensive soft tissue injury with periosteal stripping and bony exposure. This is usually associated with massive contamination.
Type III C	:	Open fracture associated with arterial injury requiring repair.

Gustilo RB, Mendoza RM, Williams DN: Problems in the management of type III (severe) open fractures: A new classification of type III open fractures. *J Trauma* 1984;24(8): 742-46.

- *Inspection:* This will reveal associated skin lacerations, which should raise the suspicion of a local tendon injury. A divided tendon end may be visible in the wound although they can be confused with deep fascia, periosteum or joint capsule. The distal limb may adopt a posture suggestive of a tendon injury (e.g. middle finger droop compared to the remaining fingers due to extensor tendon laceration). However, this should not be relied upon, as the abnormal position also relies upon gravity or an unopposed muscle action, which may be inhibited by pain.
- *Palpation:* It is not often of use in assessment of muscle and tendon injuries. However, closed tendon ruptures can be indicated by tenderness (e.g. rupture of the long head of biceps) and defect (e.g. tendo Achilles rupture).
- *Movement:* It is clearly important and involves actively testing (both for movement and power) the muscle or tendon that is suspected of damage. Care must be taken to isolate the muscle or tendon to be tested to ensure that other groups are not working (e.g. FDS and FDP in the hand).
- *Special clinical tests:* These are usually not required, as the next step having identified the lesion is to formally explore the area.
- *USS/MRI:* If there is any doubt as to whether an injury is present or not, especially in closed injuries, then ultrasound or magnetic resonance imaging can help.

Compartment Syndrome

A compartment syndrome may develop following any form of injury and does not require an underlying fracture to be present. It is increasingly associated with high levels of tissue injury and is thus more commonly associated with open rather than closed fractures.

Compartment syndrome occurs where the pressure within the affected muscle compartment rises above the capillary perfusion pressure of that compartment. This may only be around 30 to 50 mm Hg (i.e. well below systolic blood pressure). Not all compartments within the injured limb need be affected. There are only two classical findings in compartment syndrome: pain that is out of proportion to that expected for the injury (e.g. whole calf rather than at the fracture site), which often requires excessive analgesia, and pain on passive stretching of the affected muscle compartment.

- *Inspection:* This rarely reveals much to indicate a compartment syndrome. There may be some swelling, although this is often limited by the dense fascia that surrounds the compartment. Any bandaging, plasters or splints should be checked prior to any further examination for tightness, as they can mimic a compartment syndrome. Release of these (from top to bottom the whole way down to skin) should alleviate symptoms related to any tightness very quickly.
- *Palpation:* It is again relatively unrewarding as tenseness of the compartment is not always present.
- *Movement:* Involving stretching of the affected muscles is extremely painful. The patient will try to avoid active movement and minimal passive stretching of the affected compartment will be very painful.
- *Neurological and vascular examination:* These may be entirely normal and abnormal findings only tend to develop late.
- *Simple compartment pressure:* Monitors are now available and can be extremely useful in the situation where the patient is unconscious (e.g. in intensive care). They can also help where local anesthetic nerve blocks have been used (e.g. sciatic nerve block following tibial nailing).

Vascular

Vascular injuries can occur in isolation due to penetrating trauma. However, they are more commonly associated with local bony injuries, by penetration or entrapment in fracture fragments or joint dislocations.

- *Inspection*: The limb may appear pale. There may not be a large amount of blood loss or hematoma, as the vessel may be compressed by the fracture rather than divided. If there is external bleeding the wound should not be probed but external pressure applied. Other injuries may also be apparent.
- *Palpation:* It may reveal the limb to be cool. The peripheral pulses may be absent although this is not always the case. The opposite side should be checked for comparison and any difference should be treated as significant. Capillary refill may also be delayed, but can be normal where there is a good collateral circulation (e.g. around the elbow in brachial artery injuries).
- *Movement:* The patient may be unwilling to move the limb due to pain in the affected limb, although complete paralysis is a very late sign.
- *Neurological examination:* This may be unremarkable initially. Later the patient will develop paresthesia with associated reduction in sensation. As indicated above, muscle power is usually preserved until late.
- *Simple investigations:* These include the use of hand-held Doppler probes. More sophisticated Duplex ultrasound scans or more commonly, the "gold standard" arteriogram will identify the lesion.

Nerves

In a similar fashion to vessels and tendons, nerves can also be injured in an open or closed fashion. Open injuries should raise the suspicion of an underlying nerve injury. Closed injuries are often associated with nerve injuries (e.g. fibula neck fracture with common peroneal nerve palsy).

- *Inspection:* This may indicate an abnormal posture of the limb (e.g. foot drop in common peroneal nerve injury). Associated fractures or lacerations may be evident.
- *Palpation:* It will reveal little apart from associated injuries.
- *Movement* of the relevant muscle groups may be affected. However, localized pain may make examination difficult.
- *Neurological examination:* It should be conducted carefully. The usual steps of tone, power, reflexes, sensation and coordination should be sought, although may need to be modified in the light of other injuries. The aim of examination should be to isolate whether a patient has a spinal or peripheral nerve lesion, followed by determining the level or nerve affected. It should be remembered that more than one lesion might be present (e.g. brachial plexus injury) or that the lesion may not be complete.
- *Special investigations* such as nerve conduction studies and EMG's are usually unhelpful in the acute situation, as changes take several days to weeks to become established.

Systemic

In addition to the local injuries indicated above several systemic problems can occur as a result of musculoskeletal trauma.

In the acute situation the assessment and treatment of all of these (e.g. hypovolemia, crush syndrome) are well covered in the ATLS® course. Some secondary problems can occur after the admission of the patient (e.g. fat embolism, ARDS, DVT, and PE). Occurrence of most of these problems is minimized by adequately assessing and treating the patient in the first few hours after admission following trauma.

SPECIAL NOTE

Chest Injuries

Life-threatening chest injuries are:

- Airway obstruction
- Tension pneumothorax
- Open pneumothorax
- Massive hemothorax
- Flail chest
- Cardiac tamponade

Other potentially life-threatening injuries are:

- Pulmonary contusion
- Myocardial contusion
- Tear of major vessels
- Traumatic rupture of diaphragm **(Fig. 2.1)**
- Tracheobronchial tree injury
- Esophageal trauma.

Tension Pneumothorax (Fig. 2.2)

It should be diagnosed clinically and should not wait for X-ray to confirm. This results from one-way air leak from lung or chest trauma inside or outside. Patient presents with acute distress, difficulty in breathing, tachycardia, hypotension, tracheal shift to opposite side, decreased chest movements, hyperresonant note on percussion, distended neck veins, obliteration of liver dullness on right side and cyanosis in late stages.

Inserting a wide-bore needle in the 2nd intercostal space in the midclavicular line does immediate decompression. Definitive treatment is by inserting a chest tube intercostal drainage (ICD) in the 5th intercostal space just anterior to midaxillary line.

Massive Hemothorax (Fig. 2.3)

This results from rapid accumulation of blood in the pleural cavity (more than 1500 ml). This is identified by signs of hypovolemic shock with absent breath sounds and a dull note on percussion on the involved side. Treated by rapid infusion of colloids, crystalloids or blood and

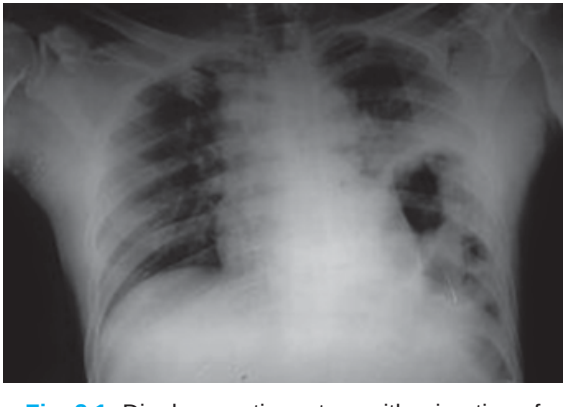


Fig. 2.1: Diaphragmatic rupture with migration of fundus of stomach into left chest cavity

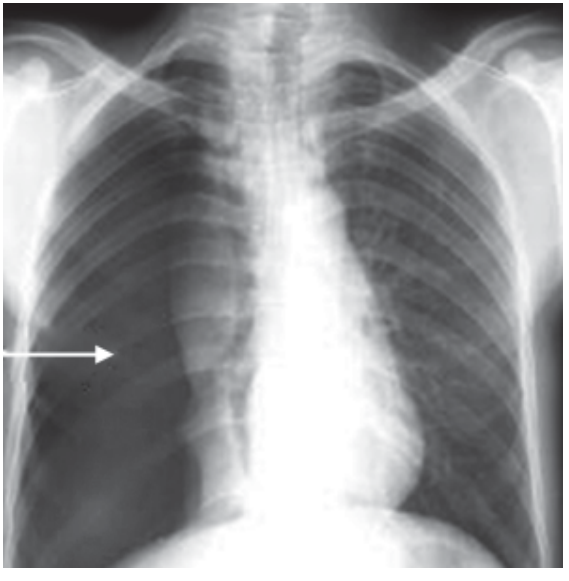


Fig. 2.2: Pneumothorax right lung with collapsed lung

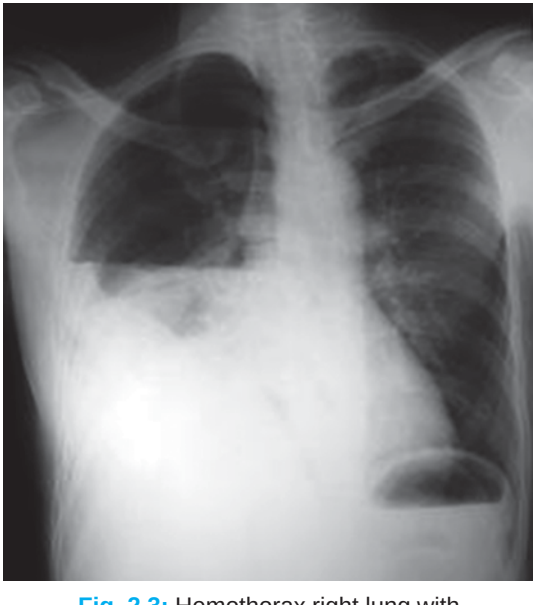


Fig. 2.3: Hemothorax right lung with horizontal fluid level

ICD. Autotransfusion from ICD may be considered in severe bleed. If the blood loss in the ICD is more than 200 ml/hour, thoracotomy may be necessary.

Flail Chest

This occurs from fracture of the chest wall resulting in a segment with no bony continuity with the rest of the bony cage. This causes paradoxical movement of the segment during inspiration and expiration. Patient will have tachypnea, tachycardia, poor air entry, abnormal chest movements and crepitus of fracture. Most of these patients have associated serious lung contusion and ventilator support may be necessary to prevent hypoxia.

Cardiac Tamponade

This is diagnosed by Deck's triad.

1. Elevated CVP with engorged neck veins
2. Decreased systolic pressure
3. Muffled heart sounds.

Electromechanical dissociation in the absence of hypovolemia suggests cardiac tamponade. Initial fluid resuscitation followed by pericardiocentesis which is done by subxiphoid route, at 45° angle, aiming towards tip of left scapula with ECG monitoring.

Pelvic Injuries

Signs of hypovolemia should be looked for, patient should be resuscitated with fluids and blood and if necessary unstable or open-book type of pelvic fractures should be stabilized with external fixator. Sometimes arteriogram and embolization or surgery may be needed to control bleeding.

Pubic ramus fractures can result in damage to urethra, which is identified by puerperal hematoma, blood at urethral meatus and inability to void urine.

Per-rectal examination can identify rectal injuries or floating prostate from urethral disruption.

Colorectal injuries may necessitate colostomy.

Sacral plexus injuries are not uncommon.

Open pelvic fractures have a high mortality due to severity and other associated injuries.

Have a definite plan for management of pelvic fractures or the approach to the fracture site may be jeopardized by external fixator application (pin tract infection) or suprapubic cystostomy (for Phannelstein's incision).

3 CHAPTER Examination of Patient with Bone and Joint Injuries

Examination should begin as discussed in the **A**dvanced **T**rauma **L**ife **S**upport (ATLS) protocol by *initial assessment and resuscitation* in previous chapter. Once the patient is stable, history and specific examination of the injured part can be done.

HISTORY

Mechanism of Injury

It is important to know the exact mechanism of injury to identify the nature of fracture, ligamentous injuries and associated injuries.

Examples

A fall from a height can present with calcaneal fracture, but one needs to assess the spine, pelvis and long bones of leg for fracture.

Dashboard injury can present with posterior dislocation hip and one needs to assess the knee for posterior cruciate ligament injury.

Fall on outstretched hand (FOOSH injury) can present with various fractures starting from finger fracture, scaphoid fracture, wrist fracture, forearm fracture, elbow dislocation, humerus fracture, shoulder fracture/dislocation and clavicle fracture.

Sudden deceleration can produce anterior cruciate ligament tear especially in sports and games, avulsion fractures or transverse fracture patella. Twisting injury to the knee with the foot on the ground can cause collateral ligament and meniscal injury.

In case of recurrent dislocation of shoulder first episode is very important to differentiate traumatic and atraumatic dislocation.

Adult pedestrian hit by car can present with triad of:

a. Leg fracture (fracture upper end of tibia)
b. Head injury (patient thrown on bonnet of car and head smashes the wind screen)
c. Wrist fractures (patient thrown off the car lands on the ground with arm stretched).

Nightstick fracture of ulna due to assault happens in reflex, protective action of flexing the elbow to ward-off blows to the face or chest.

Throwing action in sports (cricket, base-ball, javelin, discus, etc.) can cause SLAP (superior labral anterior posterior) tear of glenoid labrum in shoulder.

Pain, Swelling, Deformity and Abnormal Movements

Pain, swelling, deformity and abnormal movements corresponding to the site of fracture.

Open Fracture

In open fracture the following history are taken into consideration:

a. Time since injury.
b. Time since the patient had last oral feed – solids/liquids, important in planning time of surgery in case of emergency surgery.
c. The site of the accident for contaminants.
   Example—(i) Farm wounds of whatever size are considered type III (Gustilo's). These

wounds may have aerobic and anaerobic organisms; hence appropriate antibiotics should be administered in addition to adequate debridement.
Example—(ii) Industrial accidents. Crush injury hand may be contaminated with grease, chemicals or other foreign materials, which need appropriate treatment.

d. High or low energy injury. In road traffic accidents speed at the time of impact gives a clue to the severity of soft tissue damage in addition to the obvious fracture. Comminuted—segmental fractures implies severe soft tissue damage.

Distal Neurovascular Symptoms

Weakness or complete loss of power, altered or absent sensation, pain out of proportion to fracture (compartment syndrome—In tibial fractures, supracondylar humerus fracture).

EXAMINATION

Look

- *Attitude of the limb:* Most of the time the fracture is obvious but a single bone fracture of forearm or leg or an undisplaced fracture may be deceptive in appearance.
  Example 1: Fracture neck of femur presents with shortening and external rotation of the lower limb **(Fig. 3.1)**.

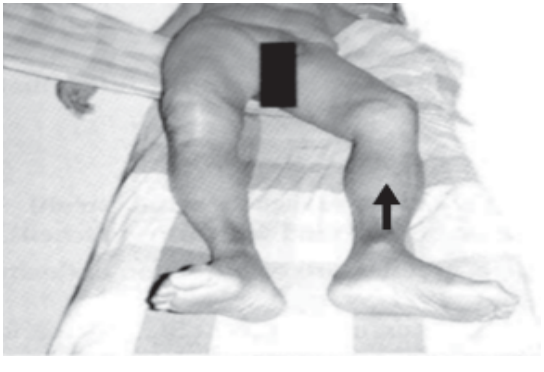


Fig 3.1: Fracture of neck of femur

  Example 2: Posterior dislocation of hip presents with flexion, adduction and internal rotation of the limb.
- *Deformity:* Abnormal appearance of the injured limb due to fracture and hematoma with change in normal alignment is noted.
- *Limb Length Discrepancy*

Feel

Local bony tenderness: Ask the patient to point the site of maximum pain. Look at the face of the patient and feel for tenderness. This is helpful in localization of the site of injury in bony bruise or fracture or ligamentous avulsions.

Muscle spasm: This can be seen and felt on feeling the fracture site and on any trivial movements.

Abnormal mobility: Most of the fractures are obvious by their abnormal mobility and it is wrong to elicit this sign as it causes pain. Patient must be made comfortable with immediate splintage and analgesics. In nonunion this sign can be elicited with or without pain.

Crepitus: Bony crepitus from movement at fracture site can be felt but should not be elicited.

Both the above signs are much appreciated at the time of splinting.

Deformity: The deformity can be due to bone itself or soft-tissue tethering and tightness, *Example:* In malunited fracture tibia, tendoachilles becomes tight causing equinus deformity.

Examination of overlying skin and soft tissue: In acute fracture the management depends on the status of soft tissue envelope. Presence of blistering or tense swelling is a contraindication for implant surgery. An abrasion over the incision site that is more than 8 hours old since the time of injury should be considered infected rather than contaminated with organisms, and surgery should be deferred until it fully heals. Splinting and elevation decreases the swelling and surgeon should wait for optimum skin condition to operate.

In chronic cases assessing the skin condition is important to avoid skin flap necrosis or wound breakdown postoperatively.

Open fracture needs assessment as described by Gustilo and Anderson to grade the severity. It is a surgical emergency and should be debrided and stabilized within 6 hours from the time of injury. Input from a plastic surgeon may be helpful in the management of wound closure. History of any infection at the time of primary treatment is very important for a treatment plan. Again a patient who had multiple operations must be assessed carefully by planning the incision to avoid skin problems. Discharging wound or sinus needs special consideration while planning surgery.

Distal neurovascular examination: Any fracture can cause neurovascular impairment. This should be checked.

Example:

Humeral shaft fracture can cause radial nerve palsy (Holstein–Lewis fracture).

Supracondylar fracture can cause injury to brachial artery, radial, median or ulnar nerves and compartment syndrome of forearm.

Upper tibia fracture is prone for compartment syndrome of leg, due to tear of anterior tibial artery as it pierces the interosseous membrane.

Fracture dislocation of the knee can produce popliteal artery injury.

Hip dislocation can cause sciatic nerve injury.

Talar dislocation can cause compression of posterior tibial artery or nerve and skin necrosis.

Examination of adjacent joints: Dislocation or ligamentous injuries to the joints should be specifically looked for as it can be easily missed.

Example: Fracture shaft of femur can be associated with hip dislocation or head or neck fracture of femur.

Move

As a quick screening, active movements of all four limbs are tested in initial assessment. Patient with fracture may find it difficult to move and fracture must be suspected if there is painful limitation. Any pain-provoking maneuver should not be done.

Adjacent joint movements should be checked in delayed union or nonunion or malunion for *post-traumatic stiffness.*

Example: Fracture lower end of femur causes tethering of quadriceps in callus and limitation of knee flexion.

Measure

Limb length discrepancy: In a displaced fracture or dislocation this is obvious. In malunion or nonunion, assessment of shortening or lengthening (e.g. femoral shaft fracture in children) is important.

Deformity: Measurement of angle of deformity in an old fracture is important. This must be expressed in three planes. Malunion or nonunion needs 3-dimensional assessment to understand the mechanical axis and to plan for correction.

SPECIFIC CONDITIONS

Compartment Syndrome

This is due to decreased perfusion from increase in interstitial pressure, impending microcirculation in a closed osseofascial compartment. Compartment syndrome can occur in closed or open fractures (17%), crush injuries, tight bandages, burns, exercise-induced, etc.

Compartment syndrome is characterized by severe pain out of proportion to the fracture; tense tender swelling; passive stretching of the involved muscle group causes pain; pins and needles and numbness due to nerve involvement, and rarely absent pulse. Symptoms are due to ischemia of muscles and nerves. The differential pressure of 20 mm less than diastolic pressure in the compartment is diagnostic of impending compartment syndrome. This needs urgent fasciotomy.

In anterior tibial compartment syndrome, there is pain along the anterior compartment of the leg with swelling, tenderness and flexing the toes causes increasing pain. Numbness in deep peroneal nerve distribution, that is first web space can be present.

The posterior tibial compartment syndrome can be of deep posterior compartment or superficial posterior compartment or involvement of both. This causes severe pain in the calf, swelling, pain on passive extension, numbness in the posterior tibial nerve distribution (deep compartment) and along sural nerve distribution (superficial compartment).

The peroneal compartment syndrome causes pain, swelling on the lateral aspect of the leg, stretch pain and numbness along the superficial peroneal nerve distribution, that is dorsolateral aspect of foot and lower leg.

Volkmann's ischemic contracture of forearm affects the flexor muscles and sometimes extensor muscles due to compartment syndrome. Tight long finger flexors due to ischemic contracture produce a tenodesis effect, extension of wrist results in finger flexion, flexion of the wrist results in extension of fingers. This is called Volkmann's sign.

In established case, contracture of muscles causes deformity like claw toes and pes cavus in the feet and claw hand with flexion contracture of the wrist.

Fat Embolism

This happens in long bone fractures due to abnormal movement at fracture site, can also be from medullary reaming for nailing. Presents with altered sensorium, chest pain, breathlessness, fever, petechial hemorrhages in the upper half of the body and subconjunctival hemorrhage. The oxygen saturation drops and can be checked with pulse oximeter to know the extent of hypoxia.

Adult Respiratory Distress Syndrome (ARDS)

It is a syndrome of acute, diffuse infiltrative lung lesions accompanied by severe arterial hypoxemia due to an overwhelming systemic inflammatory response.

ARDS has the following four findings in a patient with acute respiratory failure:

a. Severe hypoxemia, refractory to oxygen therapy. The PO_2 is less than 75 percent in patients receiving >50 percent oxygen.
b. Diffuse fluffy pulmonary infiltrates involving both lungs.
c. The absence of increased pulmonary capillary hydrostatic pressure (presence indicates a cardiogenic cause).
d. The lung demonstrates diffuse alveolar damage and generally becomes stiff with reduced lung-thoracic compliance.

ARDS commonly develops in the following conditions—polytrauma, multiple fractures, lung contusion, fat embolism, prolonged hypotension, sepsis, gastric aspiration, drug overdose and massive blood transfusion.

The mortality rate is 50 to 60 percent.

Deep Vein Thrombosis and Pulmonary Embolism

Orthopedic surgeries in the lower limb, especially hip/knee replacement or fracture neck of femur patients are prone for deep vein thrombosis. High-risk patients are those with previous history of deep vein thrombosis, obesity, immobilization, pregnancy and women on oral contraceptive pills, malignancy, varicose veins, hypercoagulable states and congestive cardiac failure. Thrombosis of calf veins, popliteal veins or femoral veins result in sudden onset pain and swelling of the lower limb, low grade fever, calf tenderness, muscles that contain thrombosed veins becomes hard and tender, and Homan's

sign (stretch pain in calf muscle) can be positive. A large swollen limb that is made pale by tense edema is called phlegmasia alba dolens or milk leg. When venous thrombosis blocks all main proximal veins the skin become congested and blue, and is called phlegmasia cerulea dolens. The incidence of clinical deep vein thrombosis is less than that confirmed with venogram and Doppler ultrasound scan.

The complications of deep vein thrombosis are pulmonary embolism which may be fatal and postphlebitic syndrome (hyperpigmentation with swelling of lower leg with varicose eczema and prone to venous ulcers) due to damage of the venous valves.

Pulmonary embolism can be massive or minor. It manifests with sudden onset chest pain, difficulty in breathing, tachypnea, pleuritic crepitation, hemoptysis, engorged neck veins, tachycardia, ECG changes and sometimes sudden collapse. Massive pulmonary embolism can be fatal.

4 CHAPTER Examination of Neuromuscular Disease

Many of the common neuromuscular diseases that require orthopedic assessment and treatment manifest in childhood. These include cerebral palsy, myelodysplasia (spina bifida) and poliomyelitis.

As with any group of disorders, a systematic approach is required to evaluate, diagnose and treat these conditions.

HISTORY

Birth History and Milestones

Cerebral palsy is often associated with prematurity and low birth weight. The birth is often difficult, and the child requires resuscitation and ventilation. The abnormality in spina bifida is usually obvious, but may be subtle involving lumbosacral skin changes in isolation. Delay in the normal motor milestones is often the first sign of an underlying neuromuscular disorder. These include head control at 3 months, sitting balance at 6 months, standing at 10 months and walking at 12 months.

Presenting Complaint

This usually involves delay or loss of motor skills including sitting, standing or walking. There are often associated delays in speech and language development and generalized medical conditions including siezures. Abnormal patterns of walking, loss of walking ability and disorders of balance may be associated with the onset of neurological disease. The time of onset and progression of the condition is important to differentiate between congenital/developmental and acquired conditions.

Assessment of Activities of Daily Living

This includes verbal and non-verbal communication ability, personal hygiene, dressing, mobility and recreation.

Family History

Family history of gait abnormalities which is relevant in muscular dystrophies and has important implications for future pregnancies. There is also an undetected chromosomal anomaly which predisposes siblings to cerebral palsy.

Past History

Immunization, previous illness, treatment and current nonskeletal conditions.

Treatment History

Previous treatment may have a significant effect on the current examination and subsequent management. This includes physiotherapy, orthotic and surgical intervention.

Knowing patient's or parents' expectation.

EXAMINATION

These children require a general examination in addition to a specific musculoskeletal assessment.

General Examination

a. Walking aids or splints: Use of rollator or frame, wheelchair, stick or splints (AFO, KAFO, DAFO) suggests the available motor power **(Fig. 4.1)**.
b. Look for truncal asymmetry, dysmorphic facies, contractures or wasting of upper or lower limbs, use of incontinence pads/nappies (in spina bifida).

UPPER LIMB

In the upper limb the various deformities may happen due to spastic hand, Erb's palsy, Volkmann's ischemic contracture or totally flail limb.

Look

In the spastic upper limb, there may be internal rotation and adduction deformity of the shoulder, flexion of the elbow, pronation of the forearm, flexion at wrist and MCPJ, and thumb in palm deformity affecting palmar hygiene.

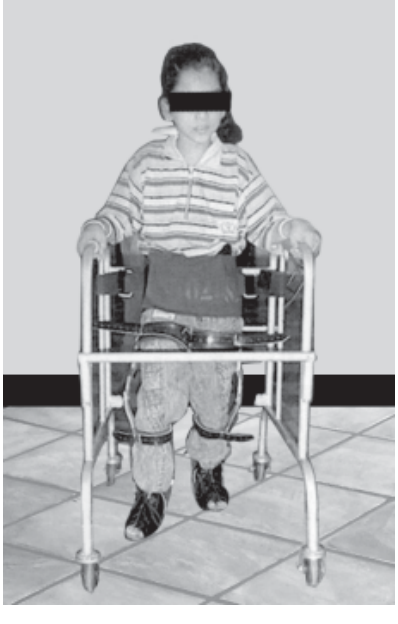


Fig. 4.1: Walking aid—child with rollator

Feel

Feel for muscle tone and power, tenderness and distal pulses.

Differentiate between upper motor neuron (UMN) and lower motor neuron (LMN) lesion.

	UMN lesion		*LMN lesion*
1.	Hypertonia of muscle-rigidity	—	Hypotonia and fasciculations
2.	Exaggerated deep reflexes	—	Absent deep reflexes
3.	Babinski's sign–positive Upgoing plantar reflex	—	Absent

Move

Assess for fixed deformities by active and passive movements.

Function

Assess the ability to position the hand in space, behind the head and to reach the back passage. This assesses the functional range of movement of the shoulder and elbow.

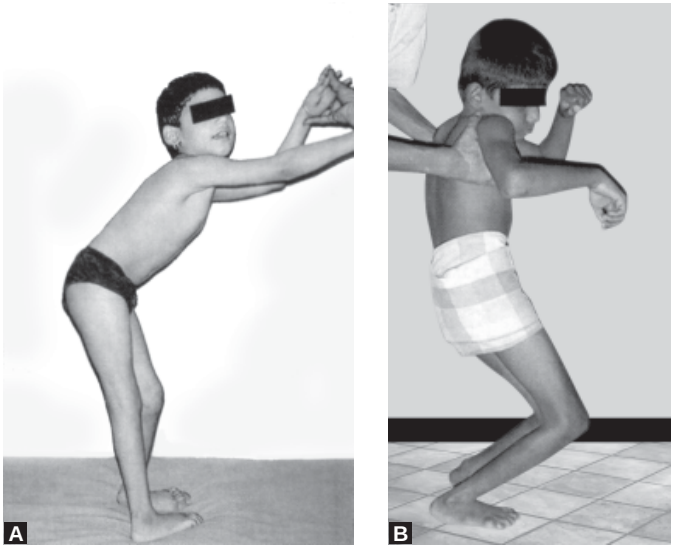
Hand function is assessed by power grip, key pinch, tip-to-tip pinch and stereognosis.

LOWER LIMB

In the lower limb the deformity may involve contractures at the ankle, knee and hip. There may be an associated gait abnormality.

Look

1. Gait: Scissoring gait, hemiplegic gait, crouched gait, jump knee gait, high stepping gait, wide-based gait **(Figs 4.2A and B)**.
2. Assess an exaggerated lumbar lordosis in fixed flexion deformity of the hip, adducted hip, flexed knee or hyperextended knee, foot deformities (equinus, calcaneus, cavus, varus or valgus), and forefoot and toe deformity.



Figs 4.2A and B: Flexed hip with normal knee gait and crouched gait respectively

3. Skin and soft tissue condition:
   a. Abnormality of the buttock and lower spine (Spinal dysraphism).
   b. Scars, trophic ulcers or callosities on the foot (Spina bifida or spinal cord lesion).
   c. Muscle wasting (Polio).
   d. Calf muscle hypertrophy (Duchenne/ Becker's muscular dystrophy).

Feel

Feel for muscle tone, tenderness and assess distal pulses.

Move

Assess for fixed deformities and contractures in each region.

Hip

1. Thomas test for fixed flexion deformity of the hip.
2. Adduction contracture: It can be due to primary adductor tightness, gracilis tightness or hamstring tightness. Remember muscle crossing two joints should be tested for contracture by putting the muscles to stretch by appropriate movements in both the joints simultaneously.
   a. Phelp's gracilis test—patient is placed prone, knees flexed and hips abducted as far as possible. Each knee is then gradually extended; the hip will adduct if the gracilis is tight.
   b. Adductor tightness—with hip flexed and knee flexed, abduct the hip.
   c. Medial hamstring tightness—with hip abducted and knee flexed, knee is extended and tightness of medial hamstrings is felt by palpation.
   d. Hamstring tightness—straight leg raise test.
3. Abduction contracture: This may be due to Iliotibial-band tightness and can be tested by Ober's test. The knee is flexed with the hip in the neutral position. The knee is extended and abduction of the hip indicates a positive test **(Fig. 4.3)**.

4. Rectus femoris tightness (Duncan Ely's prone rectus test): In the prone position the knee is flexed, with a positive test the hip flexes and causes rising of the buttock **(Figs 4.4A and B)**.
5. Rotation of the hip, torsion of the tibia and the foot thigh angle are assessed using Staheli's rotational profile (discussed in Chapter 14).

Knee

1. *Knee flexion deformity* ***(Fig. 4.5)****:* The angle between the thigh and lower leg segment gives a measure of fixed flexion deformity. Bringing the patient's knee to the edge of the couch so that the thigh segment is flat on the couch and then extending the knee determines the fixed flexion deformity. A fixed flexion deformity at the hip may give an apparent flexion deformity at the knee. Popliteal angle can be measured by keeping hip and knee at 90 degrees and slowly extending the knee to measure the hamstring tightness in spastic cerebral palsy. More than 50° is pathological **(Fig. 4.6)**.
2. *Quadriceps tightness:* This can cause limitation of flexion. In isolated rectus femoris tightness flexion of the knee will be more limited with hip extended than with hip in 90° flexion.

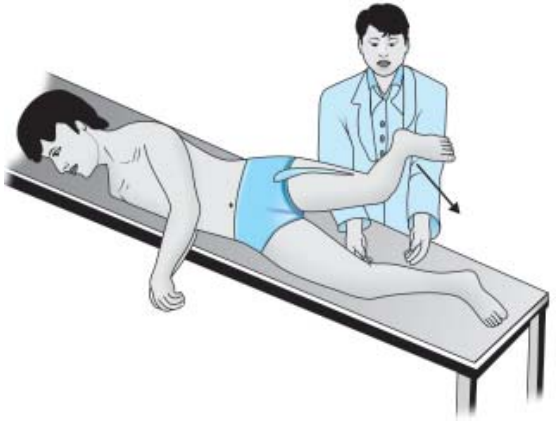


Fig. 4.3: Ober's test

Ankle

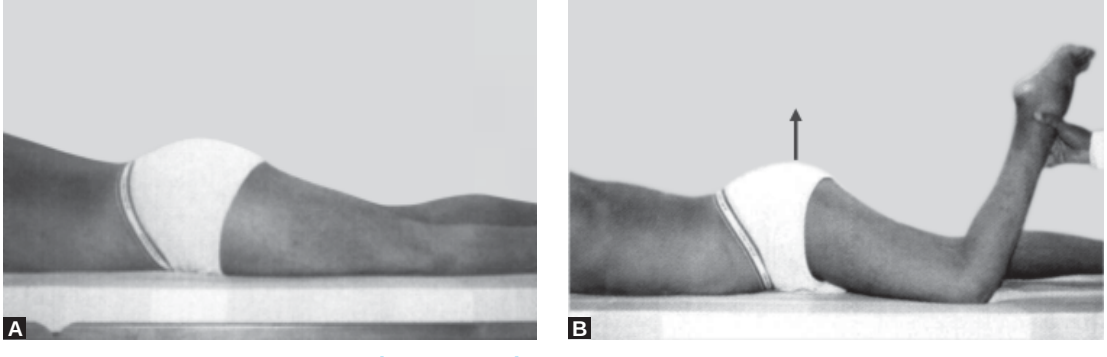
Silfverskiold's test: This differentiates between gastrocnemius or soleus contractures. Ankle movements are influenced by the position of the knee because gastrocnemius crosses both joints. In gastrocnemius contracture dorsiflexion of the ankle is limited with knee extension and more dorsiflexion of ankle is possible on knee flexion. In soleus contracture the dorsiflexion of the ankle remains the same irrespective of the position of the knee joint.

In all these patients assess the *spine* for deformity and range of motion.

SPECIAL TESTS

Gower's Sign

The patient attempts to rise from a sitting position. The sign is positive if they use their arms



Figs 4.4A and B: Prone rectus test

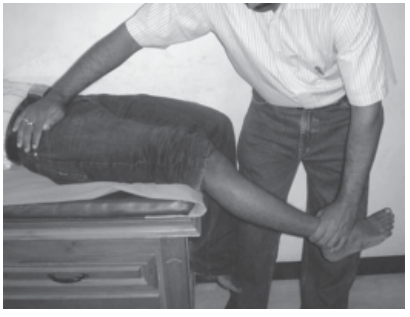


Fig. 4.5: Measurement of knee flexion deformity keeping thigh flat on the edge of the couch *(For color version, see Plate 1)*

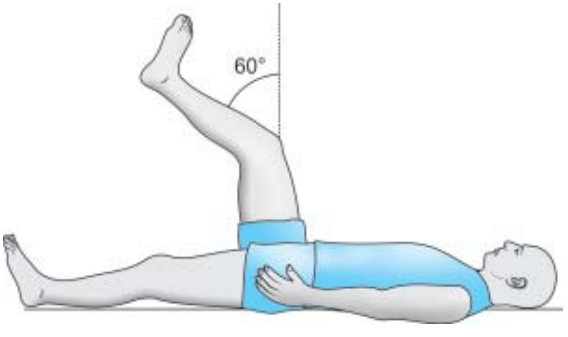


Fig. 4.6: Popliteal angle

to climb on the legs and thighs to stand. This demonstrates proximal muscle weakness and is highly suggestive of muscular dystrophy in a child. Proximal muscle weakness in elderly may be due to osteomalacia.

Meryon's Sign

The patient is lifted by holding under the arms. The sign is positive if the patient slips through because of shoulder girdle weakness.

Romberg's Sign

The patient is asked to stand with the feet together, with eyes closed. Unsteadiness indicates a posterior column lesion (Friedreich's ataxia-pes cavus). Unsteadiness with the eyes open indicates a cerebellar lesion.

Cerebral Palsy

Nonprogressive neuromuscular disorder with onset before age of 2 years, resulting from injury to immature brain. The cause is idiopathic but can be due to prenatal sickness of mother, intrauterine factors, perinatal infection (TORCH infection—Toxoplasmosis, rubella, cytomegalovirus and herpes simplex), prematurity (commonest), hypoxia and meningitis. Patient can present with diplegia (more extensive involvement of lower extremity than upper extremity), hemiplegia (involves the upper and lower extremity of same side with spasticity) or can be total body involvement. The physiologic character of presentation can be spastic (most common type with increased muscle tone and hyperreflexia with slow restricted movements because of cocontraction of agonist and antagonist), athetosis (slow writhing involuntary movement in succession), ataxia (inability to coordinate muscles for voluntary movement with unbalanced wide base gait) and mixed type.

Obstetric Palsy

Brachial plexus palsy due to stretching or contusion at the time of birth. It can be of Erb Duchenne palsy which is the most common with best prognosis. It involves lesion of C5, 6 roots affecting deltoid, rotator cuff, elbow flexors, wrist and hand dorsiflexors resulting in Waiter's tip position. Klumpke's palsy is from lesion of C8, T1 roots with deficit of wrist flexors, intrinsic hand muscles and Horner's syndrome. Total plexus lesion is from C5 to T1 with complete motor and sensory deficit and flail arm. Obstetric palsy is common in forceps delivery, large babies, shoulder dystocia, breech position and prolonged labor. Ninty percent of them resolve with maintaining passive range of movement exercises. Lack of biceps function 6 months after injury and Horner's syndrome carry a poor prognosis.

5 CHAPTER

Examination of Shoulder

The approach and sequence of examination of shoulder have changed a lot in recent years. A shoulder girdle problem can be in glenohumeral joint, acromioclavicular joint, sternoclavicular joint and scapulothoracic joint. One must get an idea of what the problem is from the age and presenting symptoms. In young adults the most common problem is instability. Instability can be defined in simple terms as symptomatic laxity. It is a spectrum of disease ranging from just pain to frank dislocation. Joint laxity without symptoms is not abnormal. In middle age the shoulder pain can be due to subacromial impingement syndrome, rotator cuff tendonitis, calcific tendonitis or frozen shoulder. In old age rotator cuff tear, glenohumeral arthritis and secondaries in proximal humerus should be considered. At any age infection like tuberculosis (caries sicca type) can happen. In elderly patient severe rotator cuff arthropathy can present with destruction of joint with bony debris, this condition is called Milwaukee shoulder. Neuropathic shoulder can occur in diabetic patients and in syringomyelia.

HISTORY

Age, Occupation and Dominant Hand

In any upper limb problem these three parameters decide the management.

Pain

Onset, duration, site and nature of pain. Patient refers most of the shoulder pain to the upper arm (deltoid region) by using the palm of other hand. Some patients present with scapular area pain in rotator cuff problem. Patients presenting with pain over the supraclavicular area and along the side of the neck may be having referred pain from a neck problem. Radicular pain (brachialgia) radiates along the nerve distribution and can extend below the elbow up to the fingers. In acromioclavicular joint pain it is well localized and the patient often points with a finger over the joint **(Fig. 5.1)**. A history of aggravating or relieving factors should be asked for. Rest pain, night pain and inability to lie on the affected side indicate the severity and nature of problem. Night pain is frequently present with rotator cuff disease, glenohumeral arthritis and frozen shoulder. Sudden acute excruciating pain without trauma may be due to acute calcific tendinitis. Pain along the medial aspect of scapula can be due to

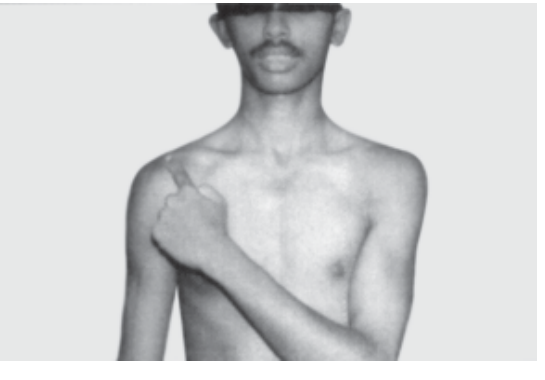


Fig. 5.1: Pointing site of pain in acromioclavicular joint problem

trapezius myofascitis or spine disease. Patient with full pain free range of movement of shoulder joint with no clinical abnormality may have referred pain from neck or lesion in and under the diaphragm or from heart disease in case of left shoulder pain.

Limitation of Activities

What stops the patient from doing the day-to-day activities like feeding, shaving, dressing, combing the hair and perineal hygiene—Is it pain or stiffness or weakness must be asked. This may affect the normal work, hobbies or lifting heavy weights.

Swelling

Diffuse swelling on top of the shoulder can be subacromial effusion, which can be differentiated from shoulder joint effusion by absence of fullness in axilla (inferior recess of the joint). Localized swelling on top of the shoulder can be due to acromioclavicular dislocation.

Stiffness

Patient presents with inability to lift the arm up, difficulty to reach the things on the shelf, inability to comb the hair, difficulty in fastening the brassiere on the back and difficulty to reach the buttocks. It may be caused by primary or secondary frozen shoulder, post-traumatic stiffness—shoulder hand syndrome, osteoarthritis, rheumatoid arthritis and rotator cuff arthropathy.

Weakness

Weakness around the shoulder can be due to intrinsic problems with the rotator cuff like partial or complete tear or neuromuscular problems like cervical radiculopathy, brachial plexus injuries, entrapment of suprascapular nerve or muscular dystrophy. In patients with suspected cuff pathology, being the most common, it is important whether the shoulder weakness followed a single traumatic event suggesting an acute tear or was a gradual onset indicative of an attrition rupture most likely secondary to impingement or intrinsic cuff degeneration. If weakness follows an injury to the shoulder then it is important to consider neurological injury to the brachial plexus, particularly the axillary or suprascapular nerves. Patient often describe the shoulder as being weak or stiff and the movement limited by pain. It is important to abolish pain by local anesthetic injection before assessing power. History of neck pain and stiffness may help in identifying patients with weakness secondary to nerve root compromise. Brachial plexus injuries are the result of violent blunt trauma to the head and neck, penetrating injuries to the posterior triangle of the neck or both injuries and should be fairly obvious. A family history of shoulder weakness occurring bilaterally starting in early adulthood and associated with facial weakness is typical of facio scapular humeral dystrophy. Patients complain of instability, inability to maintain the arms in an elevated position for a long time. Suprascapular nerve entrapment is associated with a diffuse posterolateral shoulder pain and weakness of abduction and external rotation. Confirmation requires EMG.

Instability

Feeling of joint coming out in certain positions, previous dislocations and the nature of first dislocation must be recorded. Sudden jerk or dead arm sensation on raising the arm in racquet sports due to transient subluxation causing numbness and tingling can be a presenting feature. Any instability 3 points to be noted are the degree (subluxation/dislocation), onset (traumatic, atraumatic, overuse) and direction (anterior, posterior, multidirectional). The most common glenohumeral instability being anterior and unidirectional.

Catch or Pseudolocking

This can be a symptom of instability, labral tears or from loose bodies.

Deformity

This can be from acromioclavicular dislocation, or fracture of clavicle, Sprengel's deformity or pseudoarthrosis of clavicle or unreduced shoulder dislocations or sequelae of obstetric palsy.

Miscellaneous

Involvement of other joints, neck pain with radiation to the arms along the dermatome, loss of weight, loss of appetite and constitutional symptoms.

Other relevant history must be recorded including patient's expectation **(Table 5.1)**.

CLINICAL EXAMINATION

Shoulder examination should conform to the following sequence:

1. General examination
2. Rotator cuff strength tests
3. Impingement tests
4. Biceps tests
5. Acromioclavicular tests
6. Instability tests.

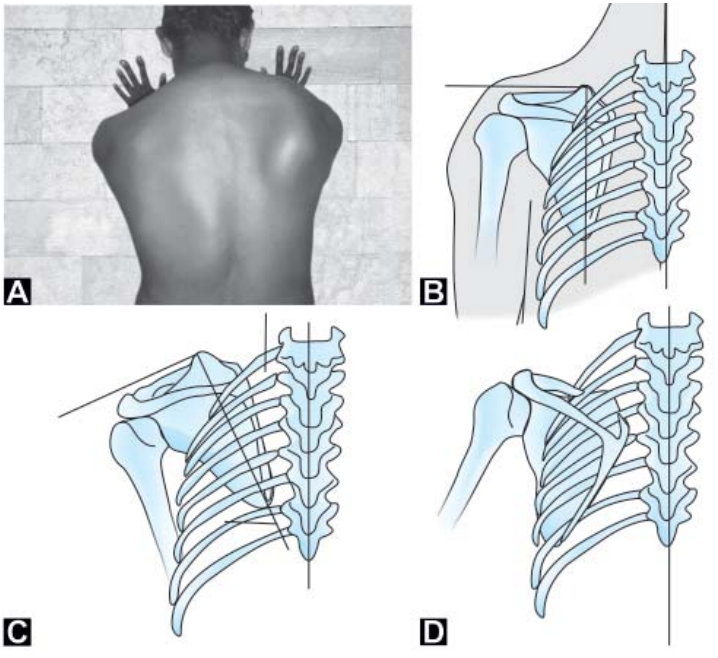
Look

Expose the patient up to waist with the patient standing or sitting. Difficulty in undressing can be noted. Patient takes off the tops on the unaffected side first and then the affected side. Look for attitude, muscle wasting, deformities— winging of scapula, and swelling. Inspect from front for shoulder symmetry, fullness of supraclavicular area, any scars, prominent acromioclavicular joint, head and neck alignment and arm position, wasting of deltoid or small muscles of the hand.

Inspect from back for any wasting of spinati. It is difficult to assess minimal wasting of supraspinatus because of overlying trapezius muscle. The most common cause of infraspinatus wasting is a rotator cuff tear. Look for winging of scapula. With paralysis of serratus anterior scapula tends to migrate proximally and its inferior angle moves medially. In contrast if trapezius is paralysed the scapula migrates downwards with inferior angle moving laterally **(Figs 5.2A to D)**. Asking the patient to push against a wall will elicit winging. Mild winging is often secondary to intrinsic glenohumeral problem like instability. Severe winging is most commonly neurologic injury. Ask the patient to raise the arm sideways up and above the shoulder. This abduction movement will give a clue to the range of abduction, any painful arc (patient may describe the feeling of pain at one particular range usually from 60° to 120°) and also look for the scapulothoracic rhythm. With normal abduction the scapula glides smoothly. In rotator cuff tear or joint pathology there will be abnormal scapulothoracic rhythm. The scapula starts to move early in abduction.

Table 5.1: Typical shoulder disorders in various age-groups

Newborn/infant	*Adolescent/young adult*	*Older adult*
Clavicular fracture	Traumatic instability	Partial tear of the rotator cuff
Torticollis	Acromioclavicular-joint separation	Complete tear of the rotator cuff
Septic arthritis	Clavicular fracture	Arthritis of the acromioclavicular joint
Osteomyelitis	Dislocation of the sternoclavicular joint	Fracture of the proximal humerus
Sprengel deformity	Tenosynovitis	Calcific tendinitis
Klippel-Feil syndrome	Atraumatic instability	Subacromial bursitis
Cleidocranial dysostosis	Subacromial bursitis	



Figs 5.2A to D: Different forms of winged scapula: (A) Winging of right scapula; (B) Normal position of the scapula; (C) Paralysis of the serratus anterior; the scapula migrates superiorly and medially; (D) Paralysis of the trapezius; the scapula migrates inferiorly and laterally *(For color version Fig. 5.2A, see Plate 2)*

The rhythm disturbance is well noted on bringing the arm down from full abduction when we can see rippling movement of scapula with asymmetry.

Swelling which is horseshoe shaped around the acromion is classical of subacromial bursitis. Ganglion cyst of acromioclavicular joint can arise from ACJ arthritis or massive rotator cuff tear. Fluid filled sac due to rotator cuff massive tear can happen in rotator cuff arthropathy (Milwalkee shoulder) or in rheumatoid arthritis or in exudative joint tuberculosis.

Feel

Feel for warmth, tenderness, swelling or irregularity in soft tissue or bony contour. Standing at the back of the sitting patient start from sternoclavicular joint palpate along the clavicle, acromioclavicular joint, subacromial area, angle of acromion, shoulder joint line, coracoid process, biceps tendon in bicipital groove **(Fig. 5.3)**, spine of scapula and tenderness along cervical spine. The biceps tendon is well felt by grasping the patient's elbow from back with one hand and turning the arm alternately into external and internal rotation. The tendon can be felt slipping under the fingertips above and lateral to coracoid process. Rotator cuff pathology causing tenderness is best felt by extension and internal rotation of the arm **(Fig. 5.4)** and feeling the greater tuberosity below and in front of the acromion (Codman's point). Also feel for any localized tenderness in the muscles or any tender palpable nodules (fibromyositis). Feel for the position of humeral head and coracoid process and along the proximal humerus. Axilla

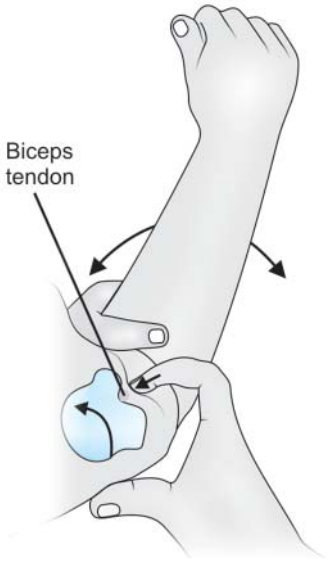


Fig. 5.3: The long tendon of the biceps is palpated in the bicipital groove with the arm in external rotation

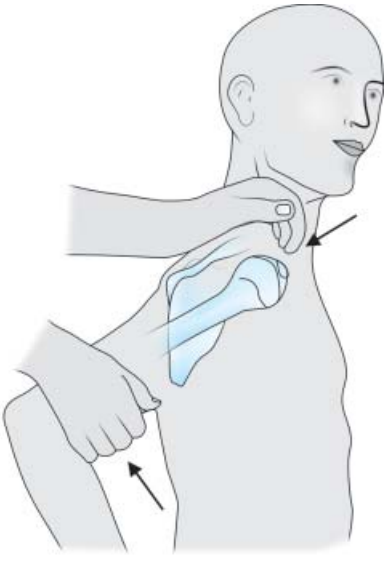


Fig. 5.4: The supraspinatus tendon and the subacromial bursa are palpated with the arm in extension

should not be forgotten. In axilla feel the humeral head, fullness of inferior recess in shoulder effusion, abnormal mass and axillary lymphadenopathy.

Move

Active abduction, forward flexion, external rotation and internal rotation should be assessed. Full range of movements indicates normal shoulder and passive movements need not be tested. Always compare with opposite normal shoulder.

Ask the patient to lift the arm sideways to know the active abduction. Abduction is lateral movement of arm to go above shoulder and hand to reach over the head (normal range 0 to 180°) **(Fig. 5.5)**.

Ask the patient to lift the arm forward; this checks active flexion (normal range 0 to 130°) and similarly asking the patient to take the arm back as far as possible checks extension (normal range 0 to 40°).

Ask the patient to clasp both hands behind the head and to take the elbow back as far as possible, this tests active functional external rotation or keep the arm by the side of the body, bend the elbow 90° and ask to turn the forearm out to assess external rotation (normal range 0 to 45°).

Ask the patient to take the hand behind the back and to reach as high as possible in the midline to assess internal rotation. This can be mentioned in terms of internal rotation to the greater trochanter, buttocks, or lumbosacral junction or to appropriate spine level (normal range if tested in 90 degrees abduction is 0 to 55°) **(Figs 5.6 to 5.8)**. Restriction of internal rotation is common in adhesive capsulitis, glenohumeral arthritis and tight posterior structures in atheletes of throwing sports. In the last condition internal rotation in 90° abduction reveals more tightness. Crossed arm adduction in forward flexion is restricted in tight posterior structures, a maneuver used to elicit acromioclavicular joint pain.

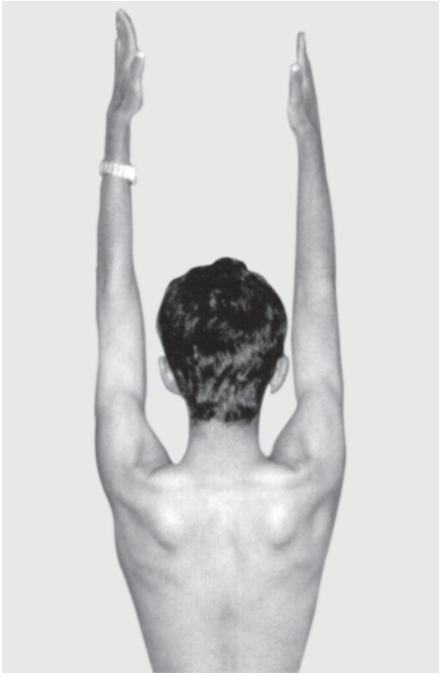


Fig. 5.5: Testing active abduction of shoulder

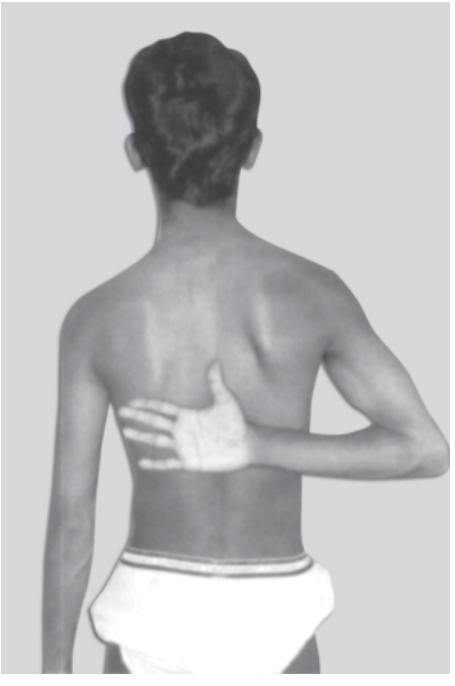


Fig. 5.6: Testing internal rotation of shoulder

Passive abduction is done in case of restricted active abduction to know the available free range of movement at the shoulder joint and to assess the painful arc. In rotator cuff tear active abduction may not be possible but passive movement may be full in early stages.

True glenohumeral movement can be assessed by fixing the scapula, press on the spine of scapula with the fingers and the thumb holding the inferior angle of scapula, the other hand can passively abduct the patient's arm. The normal glenohumeral movement is only 90° and is restricted in adhesive capsulitis **(Fig. 5.9)**.

STRENGTH TEST

It is very difficult to accurately separate the different elements by clinical testing but a gross assessment of each muscle strength can be done.

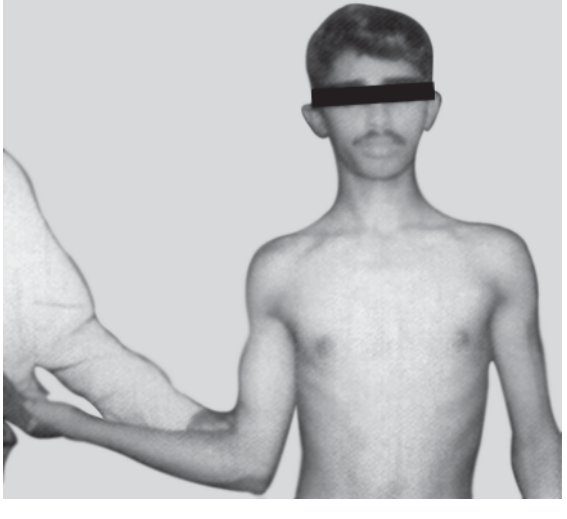


Fig. 5.7: Testing external rotation of shoulder

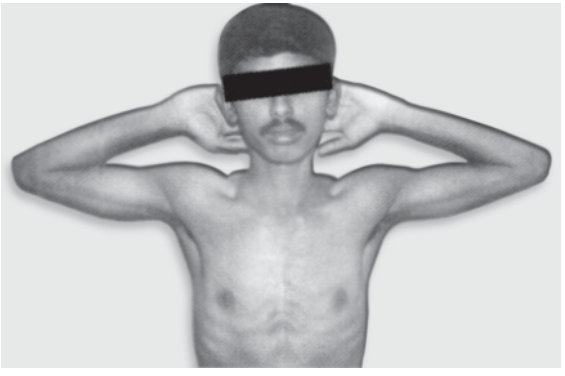


Fig. 5.8: Testing functional external rotation of shoulder

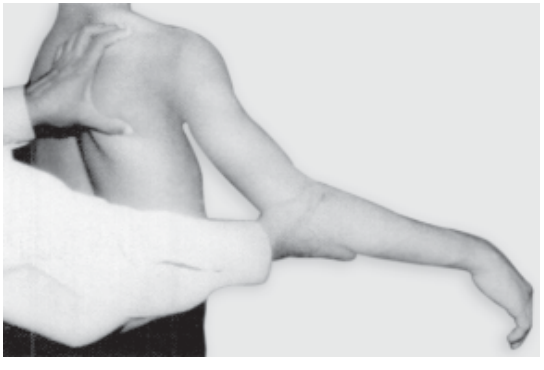


Fig. 5.9: Fixing the scapula while testing glenohumeral movement

Assess the strength of the deltoid by active resisted abduction and feel for the muscle in the lateral aspect of upper arm. This gets affected in axillary nerve palsy after dislocation of the shoulder.

To test trapezius muscle ask the patient to shrug both shoulders up and now push both shoulders down against resistance. This gets affected in spinal accessory nerve injury **(Fig. 5.10)**.

Rotator Cuff Strength Assessment

Supraspinatus Test (Jobe Test or Empty Can Sign)

Ask the patient to keep the arm forward flexed 30° and abducted 90° with thumb pointing down, fully pronated. Patient should hold the arm in the position against resistance and at the same time feel for the muscle above the spine of scapula **(Fig. 5.11)**.

Subscapularis Test

Subscapularis is the internal rotator of the shoulder and can be tested by Gerber's lift off test. Patient should internally rotate the arm to keep the hand against the buttock and try to lift off from back. Inability to do this indicates weak or torn subscapularis **(Fig. 5.12)**.

Try to push the examiner's hand back against resistance to assess the strength. It can also be grossly tested by keeping the arm adducted to the chest, elbow at 90°, forearm in midprone

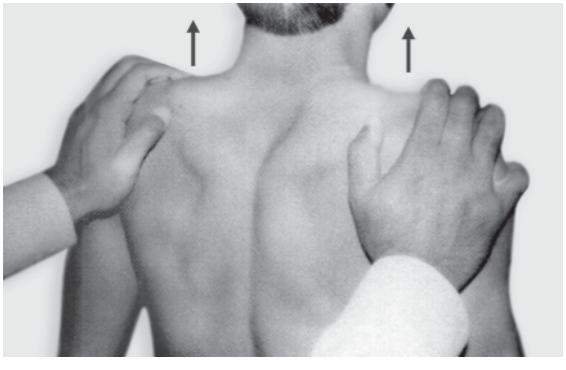


Fig. 5.10: Testing trapezius

Fig. 5.11: Supraspinatus stress test
(For color version, see Plate 2)

position and pushing the hand internally against resistance. Good power indicates normal pectoralis major and subscapularis **(Fig. 5.13)**. When there is pain or limitation of passive movement, the Gerber's test will not be possible. In that case a belly press test or Napoleon sign can be done. In this test, ask the patient to place their hands on the abdomen and examiner passively bring forward the elbows so that they are anterior to the coronal plane of the body. The patient is asked to push the hands hard into their abdomen. If either arm falls behind the coronal plane of the body, then this is suggestive of weakness of the subscapularis.

External Rotator Strength Test (Infraspinatus and Teres Minor Test)

Keep the arm by the side of the body, elbow at 90°, forearm in midprone and push the hand externally against resistance **(Fig. 5.14)**.

Active external rotation with the arm at the side of body in a 90° flexed elbow can be compared to passive external rotation. Difference in external rotation between passive and active movement indicates massive rotator cuff tear (infraspinatus) or suprascapular nerve palsy — "external rotation lag sign".

External rotation in 90° abduction is important in throwing sports and inability to externally rotate in 90° abduction or inability to hold a passive position of external rotation with the arm at 90° of abduction indicates massive rotator cuff tear (infraspinatus and teres minor) — "horn blower's sign".

Drop Arm Test

This detects whether there is any tear in the rotator cuff but is not fully reliable. First, ask the patient to fully abduct his arm and then to lower it slowly to his side. If there are tears in the rotator cuff (especially in supraspinatus), the patient will be unable to lower the arm smoothly and the arm will drop down to the side at 90°of abduction. If

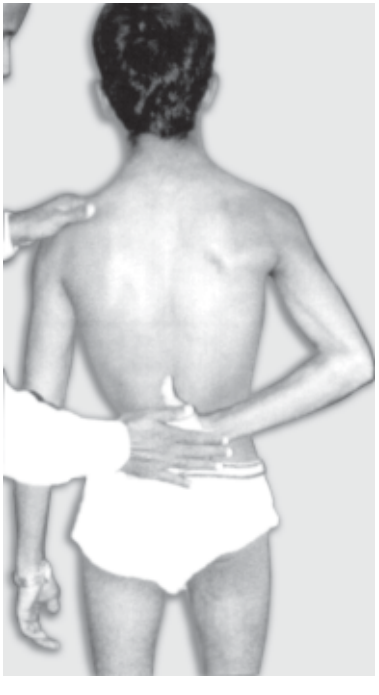


Fig. 5.12: Gerber's lift-off test

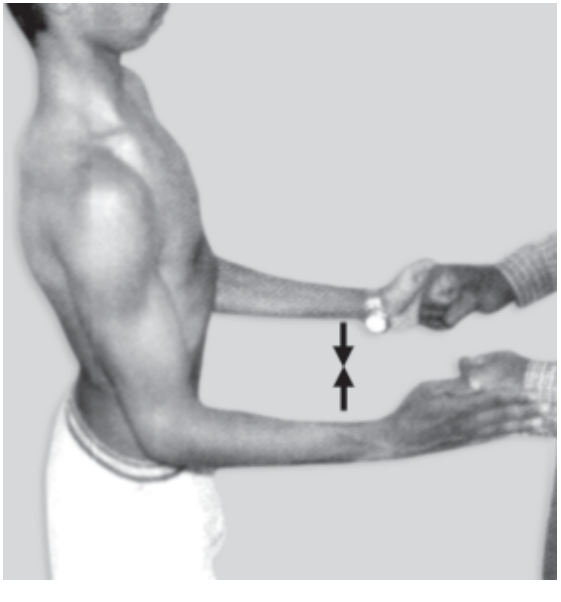


Fig. 5.13: Testing internal rotators of shoulder

patient is able to hold the arm in abduction, a gentle tap on the forearm will cause the arm to drop to the side. In complete tear of the supraspinatus the patient may not be able to initiate abduction and can do trick movements like swaying the body to the affected side for initial abduction and then with the help of deltoid can lift the arm further **(Fig. 5.15)**.

The abductor paradox—with a complete tear of supraspinatus tendon, patient will be unable to initiate abduction but if the arm is passively lifted he or she can hold it abducted using the deltoid and the remaining intact cuff **(Tables 5.2 and 5.3)**.

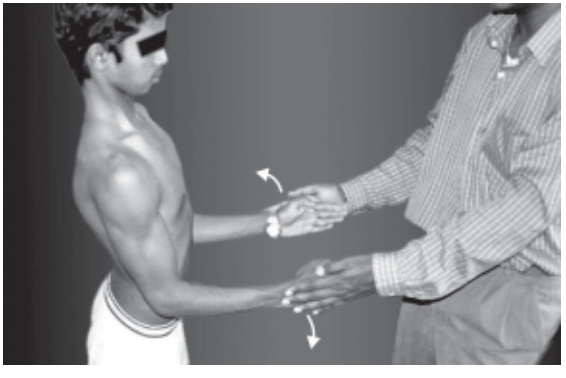


Fig. 5.14: Testing external rotators of shoulder

IMPINGEMENT TESTS

Hawkin's Test

Arm in 90° forward flexion, elbow bent to 90° with the patient relaxed using forearm as a lever internally rotate the arm supporting the elbow with one hand. Sharp catchy pain indicates impingement syndrome **(Fig. 5.16)**.

Neer's Impingement Sign

With one hand raise the affected arm in forward flexion while the other hand supports the shoulder. Forward flexion in the scapular plane can elicit pain when shoulder goes beyond 90° due to abutment (forward flexion painful arc). This causes pain due to impingement of the greater tuberosity against the coracoacromial arch and is seen in subacromial impingement syndrome (supraspinatus tendonitis, bursitis, partial tear of rotator cuff) or calcium deposition **(Fig. 5.17)**.

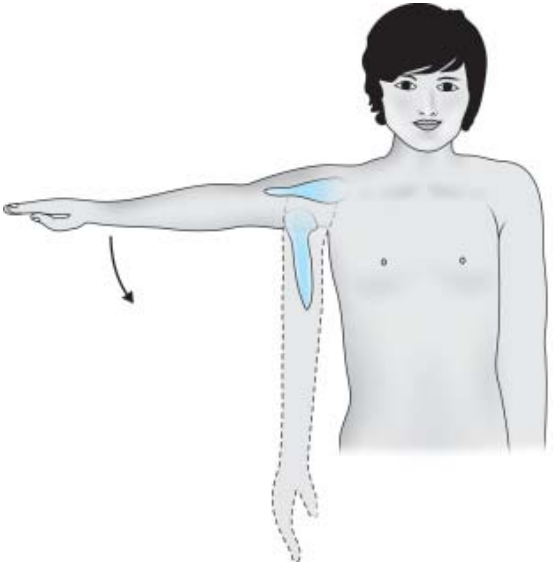


Fig. 5.15: Drop arm test

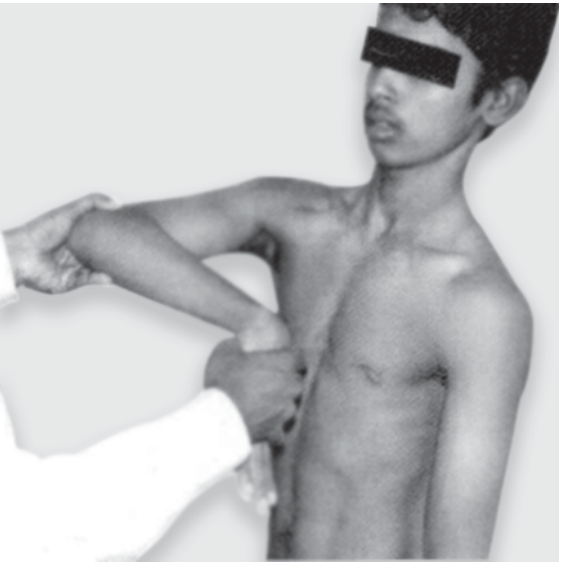


Fig. 5.16: Hawkin's test

Table 5.2: Muscle testing chart

Muscle	*Innervation*	*Myotomes*	*Technique for testing*
Trapezius	Spinal accessory	C2-C4	Patient shrugs shoulders against resistance
Sternomastoid	Spinal accessory	C2-C4	Patient turns head to one side with resistance over the opposite temporal area
Serratus anterior	Long thoracic	C5-C7	Patient pushes against a wall with an outstretched arm Scapular winging is observed
Latissimus dorsi	Thoracodorsal	C7-C8	Downward/backward pressure of the arm against resistance. Muscle palpable at inferior angle of the scapula during cough
Rhomboids	Dorsal	(C4) C5	Hands on hips pushing elbows backward against resistance
Levator scapulae	Scapular		
Subclavius	Nerve to subclavis	C5-C6	None
Teres major	Subscapular (lower)	C5-C6	Similar to latissimus dorsi; muscle palpable at lower border of the scapula
Deltoids	Axillary	C5-C6 (C7)	With arm abducted 90 degrees, downward pressure is applied. Anterior and posterior fibers may be tested in slight flexion and extension
Subscapularis	Subscapular (upper)	C5	Arm at side with elbow fixed to 90°. Examiner resists internal rotation
Supraspinatus	Suprascapular	C5 (C6)	Arm abducted against resistance (not isolated). With arm pronated and elevated 90° in the plane of scapula, downward pressure is applied
Infraspinatus	Suprascapular	C5-C6	Arm at side with elbow flexed 90°. The examiner resists external rotation
Teres minor	Axillary	C5-C6 (C7)	Same as for the infraspinatus
Pectoralis major	Medial and lateral pectoral	C5-T1	With arm flexed 30° in front of the body, the patient adducts against resistance
Pectoralis minor	Medial pectoral	C8, T1	None
Coracobrachialis	Musculocutaneous	(C4)C5-C6 (C7)	None
Biceps brachii	Musculocutaneous	(C4)C5-C6 (C7)	Flexion of the supinated forearm against resistance
Triceps	Radial	(C5) C6-C8	Resistance to extension of the elbow from varying positions of flexion

Table 5.3: Neurologic level In upper limb

Level	*Motor*	*Sensory*	*Reflex*
C5	Deltoid Biceps (partial)	Lateral deltoid	Biceps
C6	Biceps ECRL and ECRB	Thumb	Brachioradialis Biceps
C7	Triceps Wrist flexors Finger extension	Middle finger	Triceps
C8	Finger flexors	Ulnar border Little finger	—
T1	Intrinsics	Medial side Proximal part of arm	—

Dermatomes in the upper limb:
C5-lateral deltoid
C6-thumb
C7-middle finger
C8-little finger
T1-inner aspect of the proximal part of the arm

Neer's Injection Test

In patients with painful arc, inject 10 ml of 1 percent lignocaine into the subacromial area and wait for 5 minutes. If the painful arc disappears it is diagnostic of impingement syndrome.

Coracoid Impingement Sign

In coracoid impingement syndrome palpation in the region of coracoid causes discomfort. This sign is elicited by abducting up to 90° in coronal plane and maximally internally rotating the arm to cause pain—"tennis serve follow-through " position.

BICEPS TEST

Tenderness can be elicited along the groove due to inflamed bicipital sheath (but beware because it is often tender to palpate near the coracoid try to see if the tenderness moves as the shoulder and biceps moved, and therefore, is rotated).

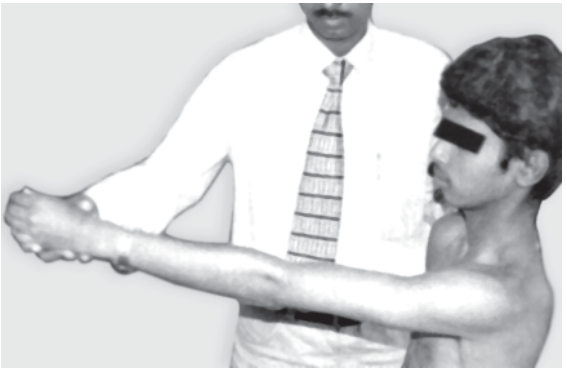


Fig. 5.17: Neer's impingement sign

Speed's Test (Palm-up Test)

Forward elevation of the upper extremity between 60 to 90° with the elbow in extension and the forearm in supination against resistance causes pain at the long head of biceps **(Fig. 5.18)**.

Yergason's Test

Do resisted supination of the hand with the elbow in flexion. Pain may be felt at the long head of biceps.

Lippman Test

Examiner palpates the bicipital groove approximately 3 cm distal to the shoulder with the patient's elbow flexed at a right angle. One can provocate biceps tendon subluxation or dislocation by palpation of relaxed muscle. This is generally painful for the patient. This can also be demonstrated with slow adduction and abduction while palpating the tendon (Gilcrest test).

ACROMIOCLAVICULAR TESTS

Patient localizes the pain at acromioclavicular joint.

Terminal Painful Arc

Pain during terminal abduction of 160° to 180°, indicates acromioclavicular joint pathology.

Fig. 5.18: Speed test to evaluate the long head of the biceps

Flexion Adduction Test

Taking the arm across the chest towards the opposite shoulder with a bent elbow can cause pain at acromioclavicular joint **(Fig. 5.19)**.

INSTABILITY TESTS

Instability is symptomatic inability to maintain the humeral head in glenoid.

Before testing ask the patient whether he can dislocate the shoulder himself, to know if there is a voluntary component.

Generalized Ligament Laxity

Assess for generalized ligament laxity as discussed in Chapter 12 (Patellofemoral joint problem).

Sulcus Sign

Patient sitting with arms relaxed by the side of the body, with the elbow flexed to 90°, give a downward axial force along the humerus by holding the elbow. The sulcus sign appears between the acromial arch and head of humerus laterally. The sulcus test demonstrates the degree of inferior capsular laxity and tests the superior glenohumeral ligament and coracohumeral ligament. Always compare with other side **(Fig. 5.20)**.

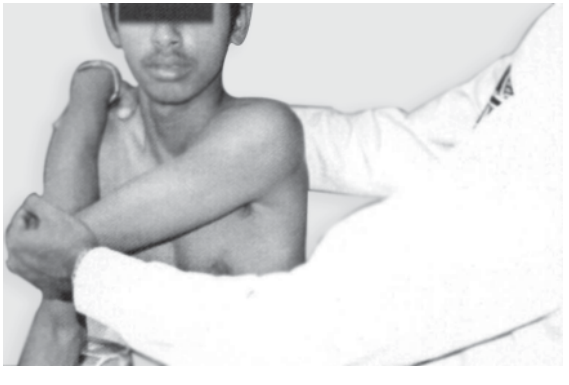


Fig. 5.19: Acromioclavicular joint stress test

Anterior Translation Test (Load and Shift Test)

Patient sitting, arm by the side of the body, patient relaxed with forearm over the lap, examiner standing from back, with one hand hold the scapula with fingers in the front of shoulder over coracoid and thumb in the back over the angle of acromion, with the other hand hold the head of humerus. Perform the anterior translation test. The amount of anterior translation is graded. Grade 1-humeral head rides up the glenoid slope but not over the rim; Grade 2-humeral head rides up and over the glenoid rim but reduces spontaneously when stress is removed; Grade 3-humeral head rides up and over the glenoid rim and remains dislocated on removal of stress. Comparison must be made to the asymptomatic contralateral side. This test can also be performed in supine position while examining the patient under anesthesia **(Fig. 5.21)**.

Although translation is assessed initially in neutral position with the arm by the side, supine load and shift testing in varying arm positions give more information of anterior and posterior glenohumeral ligamentous complex. By progressive external rotation and abduction there is less translation anteriorly, as inferior glenohumeral ligament becomes taut. Similarly by internal rotation of the arm posterior translation is diminished with intact posterior capsular structure. With arm at the side inferior restraints are superior ligamentous structures. In 90° abducted position, the primary restraint to inferior translation is the inferior glenohumeral ligament.

Apprehension Test

Arm in 90° abduction, external rotation and extension with slight forward pressure placed on proximal humerus, look at patient's face for apprehension or pain or feeling of instability. This can be done in sitting or supine position. This indicates anteroinferior instability **(Fig. 5.22)**. Pain alone in apprehension position especially in an overhead athelete is strongly suggestive of

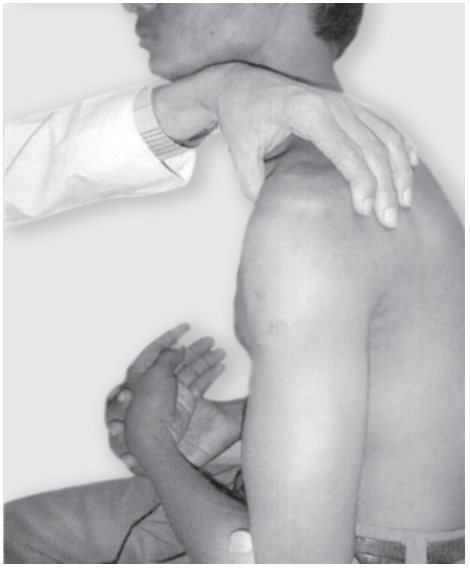


Fig. 5.20: Sulcus sign

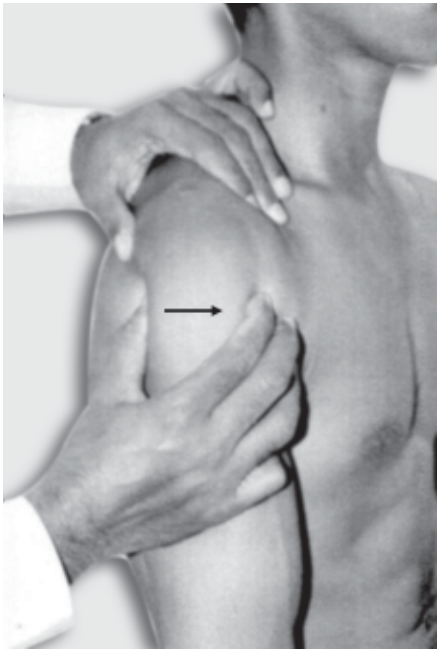


Fig. 5.21: Anterior translocation test

posterosuperior glenoid labral pathology or internal impingement—posterior glenoid labrum in contact with articular surface of rotator cuff at the anterior edge of infraspinatus.

Jobe's Relocation Test and Release Test

The apprehension test is supplemented by relocation maneuver. Patient supine, bringing the shoulder to edge of the couch perform the apprehension test as described above. Simply pushing the humeral head back with the examiner's fingers can relieve the apprehension. This permits further external rotation and extension. This relocation manoeuvre eliminates any chance of anterior subluxation. Silliman-Hawkin's release test is releasing the backward pressure of humeral head suddenly in extreme external rotation that causes rebound apprehension and sometimes dislocation. This test is best avoided in more apprehensive patients. This test can also be positive with pain from internal impingement rather than apprehension **(Fig. 5.23)**.

The apprehension test and relocation test can be positive for instability, for pain, or for both. Positive tests for instability indicate anterior instability. Positive test for pain indicates posterosuperior labral pathology or internal impingement.

Posterior Load and Shift Test

To assess posterior instability. Flex the shoulder 90°, flex the elbow fully and in slight adduction give an axial load along humerus holding elbow with one hand and neck of scapula with other hand. Feel for posterior subluxation or dislocation. Push/pull (Norwood) and posterior drawer test (Gerber) can also confirm the posterior laxity **(Fig. 5.24)**.

Young patients with instability problem can sometime present with shoulder pain due to secondary impingement (abnormal head of humerus movement and impingement against acromial arch).

NECK EXAMINATION

Neck should be examined to rule out any radiating pain to the shoulder especially in bilateral shoulder pain, pain radiating to the arm below elbow and pain predominantly over suprascapular area.

Pain in left shoulder can be a referred pain from heart and upper gastrointestinal problem. Pancoast tumor should also be considered in elderly patient with supraclavicular pain and swelling.

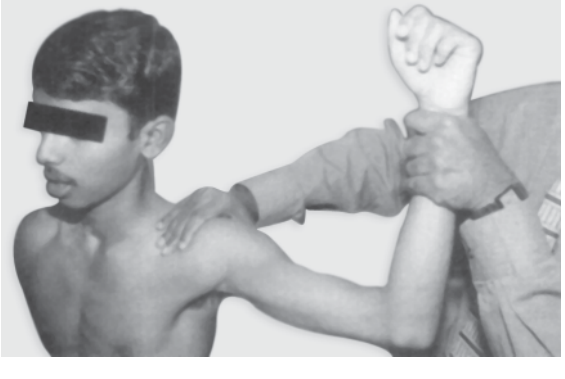


Fig. 5.22: Apprehension test

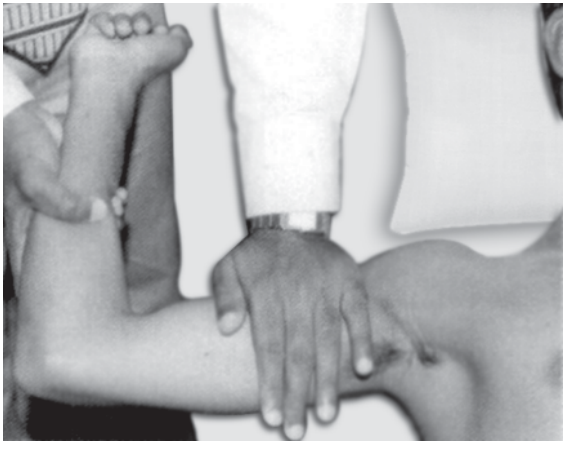


Fig. 5.23: Jobe's relocation test

Fig. 5.24: Posterior stress test

NEUROVASCULAR EXAMINATION

For completion, examination of neurological system and vascular system of the upper limb should be done.

COMMON CONDITIONS AFFECTING SHOULDER

Rotator Cuff Disease

It is a spectrum of pathology ranging from inflammation in subacromial bursa and rotator cuff to partial or complete tear and eventually rotator cuff arthropathy (secondary OA with proximal migration of humeral head). This is due to impingement of rotator cuff insertion against the coracoacromial arch. This microtraumatic process results in pain and dysfunction and is called subacromial impingement syndrome.

Bigliani has desribed 3 acromial morphologies:

- Type 1: Flat acromion;
- Type 2: Curved acromion;
- Type 3: Hooked acromion.

The type-3 acromion is most commonly associated with impingement. Anterior acromial spur and inferior osteophytes at ACJ, unfused acromial epiphysis (Os acromionale) and anterolateral overhang of coracoid process are causes of impingement.

Macrotrauma of rotator cuff leading to tear is common in elderly patients, e.g. an elderly patient with fall on outstretched hand and not able to lift the arm up with no fracture or dislocation is most likely to have cuff tear. The clinical presentation can be of pain-constant or night pain, weakness or loss of function. Physical examination will demonstrate tenderness at subacromial area, sometimes ACJ and biceps tenderness, painful abduction arc usually from 90 to 120 degrees and positive impingement signs verified by impingement tests. Rotator cuff strength tests, ACJ tests and biceps test should be done for complete assessment.

Calcific Tendinitis

Calcific deposits in the rotator cuff tendons are usually seen in fifth or sixth decade. Acute stage is characterised by severe pain, patient holding the arm to the side of the chest and not allowing even the slightest movement. Local tenderness may be present. In the chronic stage the pain is less and symptoms and signs of impingement may be present.

Bicipital Tendinitis

Biceps tendon serves as humeral head depressor. It can be affected in variety of disorders—biceps tendonitis, subluxation or dislocation of tendon and rupture of biceps tendon. Tendinitis and rupture occur almost universally as a result of impingement and rotator cuff disease. Subluxation of biceps tendon occurs with the disruption of subscapularis attachment to the lesser tuberosity as the adjacent transverse humeral ligament is disrupted. Always perform the Gerber's lift off test in suspected subluxation. The other causes of subluxation include primary rupture of transverse humeral ligament or fracture of greater or lesser tuberosity. In ruptured long head of biceps the muscle stands with a bulge in the middle of the arm—Popeye's sign.

Superior Labral Anteroposterior (SLAP) Lesion

Patient can present with pain, clicking or popping in the shoulder, common in athletes with overhead throwing sporting activities. The abnormality is at the site of origin of the long head of biceps from the superior labrum both anteriorly and posteriorly. Clinical examination may demonstrate positive Speed or Yergason's test.

O'Brien's test or active compression test is very sensitive and specific test for SLAP lesion. Ask the patient to hold the arm in 90° forward flexion, 10° adduction and full internal rotation of the arm and resist downward force of the arm. Patient should point out the site of pain to differentiate from acromioclavicular joint pain. This pain should disappear on external rotation of the arm in the same position on resistance.

Anterior slide test: With patient standing with the hands on the hips and thumbs pointing posteriorly, place examiner's one hand over the affected shoulder with the index finger over the anterior aspect of acromion. With examiner's other hand behind the patient's elbow, apply force in an anterosuperior direction. Instruct the patient to push back against this force. Sudden pain in the anterosuperior shoulder typically experienced during exercise or a palpable snap phenomenon is indicative of a SLAP lesion **(Figs 5.25 and 5.26)**.

Biceps load test can also be done to diagnose superior labral and SLAP lesions. With patient supine shoulder abducted to 120° and maximally externally rotated the forearm in maximum supination and elbow placed at 90° of flexion, forceful flexion against resistance done to elicit pain in the shoulder.

Osteoarthritis Shoulder

This can be primary OA usually with intact rotator cuff or secondary OA from trauma, cuff tear arthropathy, infection or avascular necrosis of humeral head. Patient present with pain, stiffness and clinically there is local tenderness along anterior and posterior joint line, and limitation of

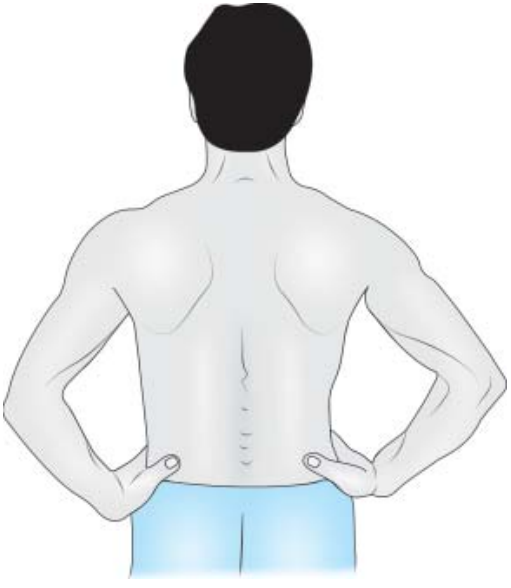


Fig. 5.25: Slide test: Position of the hands and arms for the anterior slide test

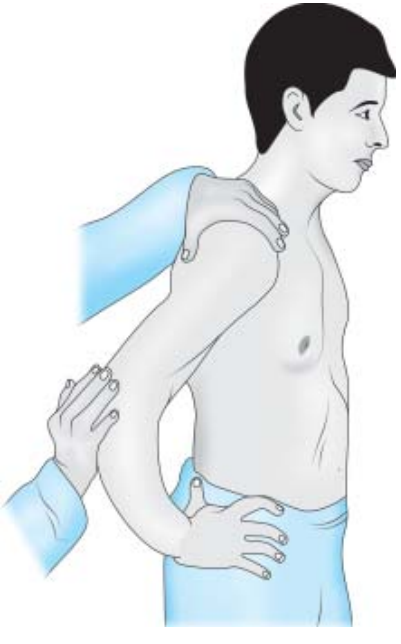


Fig. 5.26: Slide test: Application of force for the anterior slide test

glenohumeral movements. Pain is worse on recumbence as the distraction force in upright position is absent. Functional limitation of rotational movements is more noticeable than limitation of glenohumeral abduction as scapulothoracic joint compensates this movement.

Rheumatoid Arthritis

Characterized by polyarticular involvement, pain, swelling, morning stiffness and restriction of movements.

Shoulder Instability

Common in young adults and varies from pain to frank dislocation due to laxity of capsulotendinous structures. First episode must be clearly documented for mode of injury, mechanism, direction of dislocation. Number of subsequent dislocation and movement that precipitate are noted. If there is no history of trauma it is important to ask if the patient can voluntarily dislocate.

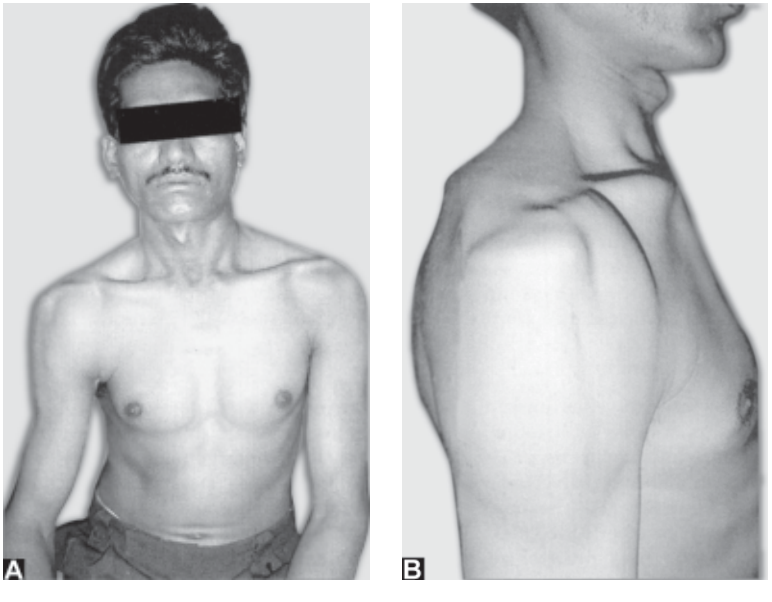
Painless clicks are quite common in shoulder especially in frozen shoulder. Painful click can arise from SLAP lesion.

Matsen described 2 acronyms:

- TUBS—Traumatic, Unilateral, Bankart's lesion and treatment is Surgery.
- AMBRII—Atraumatic, Multidirectional, Bilateral and treatment is Rehabilitation or Inferior capsular shift and closure of rotator cuff interval.

Feeling of insecurity, apprehension in certain positions, joint subluxation or dislocation, pain due to secondary impingement and dead arm syndrome (momentary subluxation) are some of the features. Stability tests must be done to identify anterior, posterior or inferior laxity.

Traumatic anterior dislocation of shoulder is characterized by loss of normal contour **(Figs 5.27A and B)**, bony mass (humeral head) in front of the shoulder, inability to internally rotate, hand not able to touch the opposite shoulder and shortening of arm segment (posterior angle of acromion to lateral



Figs 5.27A and B: Anterior dislocation of shoulder

epicondyle). Duga's sign will be positive where the patient is unable to touch the contralateral shoulder with the hand of the affected arm. Dead arm syndrome is associated with anterior subluxation.

Posterior dislocation is common in electric shock or following fits. Loss of shoulder contour with limitation of external rotation is characteristic.

Frozen Shoulder (Adhesive Capsulitis)

This disorder evolves in three stages:

Stage 1 is characterized by diffuse pain gradual in onset. Stage 2 is characterized by stiffness, affects activities of daily living with limitation of all movements more specifically forward flexion and external rotation limitation.

Stage 3 is thawing, with gradual return of motion. It is a self-limiting condition with natural history of the disease lasting for 18 months to 2 years. The cause is unknown, occurs in middle age female population but more common in diabetic patients, thyroid disorders and post surgical. Other than primary idiopathic frozen shoulder secondary causes include impingement syndrome, rotator cuff tear, calcific tendonitis and following trauma (post-traumatic stiffness). Frozen shoulder is a diagnosis of exclusion of other conditions in shoulder and is a rare condition.

Shoulder Crepitus

Crepitus can arise from subacromial, glenohumeral or scapulothoracic articulation. Common causes of crepitus about the shoulder are rotator cuff tear, glenoid labral tears, glenohumeral osteoarthritis, scapulothoracic bursal scarring or exostosis emanating from ribs or under surface of scapula.

6 CHAPTER

Examination of Elbow

Elbow joint is a complex structure consisting of three separate articulations: The humeroulnar, humeroradial and proximal radioulnar joint. Humeroulnar joint is a hinge joint allowing flexion and extension. Forearm rotation of supination and pronation is at radioulnar joint. Introduce yourself and ask the name, age, occupation and the dominant hand. The presenting complaint of any elbow problem can be of pain, stiffness, swelling, deformity, instability and neurological symptoms. Level of activities must be assessed to know the loss of function like hand to mouth, for perineal hygiene, lifting and carrying things and job-related activities. History of injury, other joint problems and other relevant medical history are important.

Various disorders can be caused by acute injuries, chronic stressors, degenerative disorders and systemic disorders. In repetitive motion clearly defined clinical syndrome like tennis elbow can happen in patients like electrician, carpenter or racquet sports. Patient's expectation should be known.

LOOK

From the Front

With both elbows fully extended, arms by side and palms facing forward look for the long axis of the upper arm and forearm which forms a lateral valgus angle called carrying angle. The physiologic range for this angle is between 10 to 15° in women and 5° in men. Look for the deformity—cubitus varus—the angle is less than 5° **(Fig. 6.1)** or cubitus valgus **(Fig. 6.2)** —the angle is more than 15°, swelling, scars, skin color, wasting of radial muscles (mobile wad-brachioradialis, extensor carpi radialis longus and brevis) or ulnar muscles of forearm, or small muscles of hand. Cubitus varus is commonly due to childhood trauma such as malunited supracondylar fracture or early physeal closure medially. Cubitus valgus is commonly due to lateral condyle nonunion or malunion or due to physeal damage.

Ask the patient to show both elbows with hands reaching the shoulder (flexion of elbow). In a flexed elbow assess for deformity and bony prominences—medial epicondyle, lateral epicondyle and tip of olecranon.

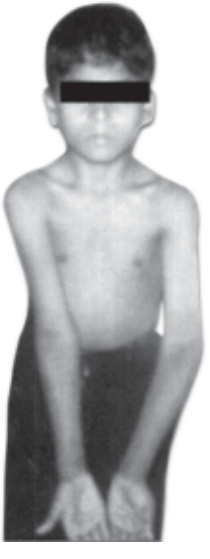


Fig. 6.1: Cubitus varus

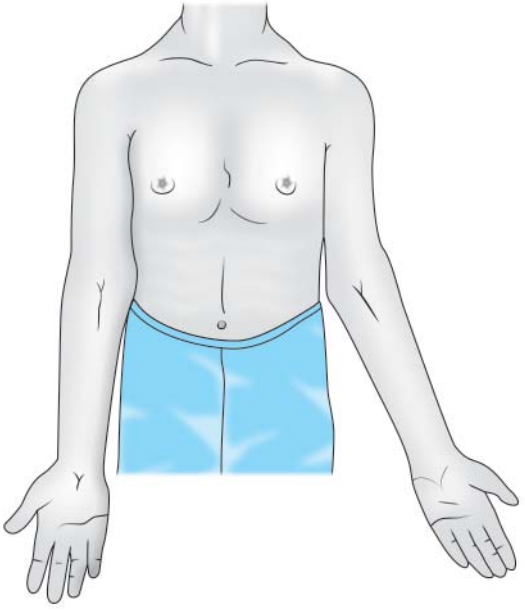


Fig. 6.2: Cubitus valgus of left arm

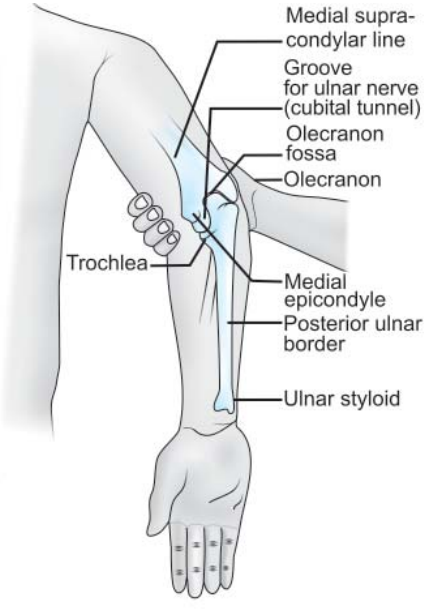


Fig. 6.3: Palpable structures in the elbow region

Swellings like olecranon bursitis (fluctuating swelling over tip of the elbow), rheumatoid nodule (a firm swelling over the olecranon and subcutaneous border of ulna) or gout (ruddy complexion) can be assessed.

From Side

Ask the patient to straighten the arms out and look tangentially from the side for limitation of terminal extension or hyperextension.

Swelling and scars should be noted.

From Back

Look for swellings, scars and abnormal bony prominences by comparing the opposite side. Fullness on either side of olecranon at the insertion of triceps can be due to effusion of the joint.

FEEL

Ask the patient for tender spot and look at the patient when you feel.

Start from the medial side **(Fig. 6.3)**. A tender medial epicondyle should be tested for pain on resisted wrist flexion in supination. This indicates medial epicondylitis (Golfer's elbow) or injury to medial epicondyle. Medial supracondylar line of humerus can be palpated superior to the epicondyle. Wrist flexors arise on this and occasionally a small bony prominence will be palpable which can compress the median nerve.

Ulnar nerve is palpated behind the medial epicondyle in the groove between medial epicondyle and olecranon for thickening and tenderness. Tinel's sign—pins and needles or sharp sensation along the ulnar nerve distribution can be elicited by gentle percussion of the nerve. It can get injured in supracondylar or epicondylar fractures. Recurrent subluxation of the ulnar nerve should be tested by assessing the stability in flexion and extension movements. Thickening of the nerve can occur in Hansen's disease and hypertrophic neuropathy.

Tenderness over the olecranon and radial head should be assessed. Radial head tenderness

is assessed by supination-pronation of the forearm and is best felt in 90° flexion of the wrist and 2.5 cm distal to lateral epicondyle and any abnormal subluxation or dislocation (radiocapitellar joint) can be felt **(Fig. 6.4)**.

Lateral epicondyle tenderness is then assessed. Lateral epicondylitis (Tennis elbow) is assessed by Cozen's test **(Fig. 6.5)**, where resisted extension of the wrist in pronation causes pain at the lateral epicondyle. Resisted extension of the middle finger causes pain at the extensor carpi radialis brevis origin (lateral epicondyle) as it is the usual pathological muscle. Testing the middle finger extension causes pain because extensor carpi radialis brevis inserts into 3rd metacarpal base. Mills test is again helpful where full volar flexion of the wrist with full extension of the elbow cause pain when there is tightness of the extensor carpi radialis brevis. Lateral supracondylar line can be palpated where extensors of wrist arise, and this bony margin can be palpated up to deltoid tuberosity.

Bony thickening should be appreciated. Synovium and effusion of the joint can be well felt in a triangular space between lateral epicondyle, olecranon and radial head.

The anterior aspect of cubital fossa can be palpated for any abnormal mass and biceps tendon. The biceps tendon, brachial artery and the median and musculocutaneous nerves pass through this region from lateral to medial.

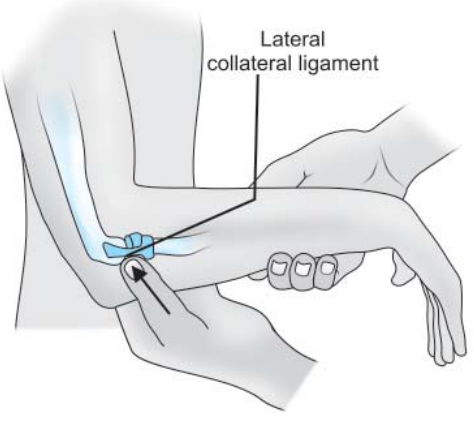


Fig. 6.4: Palpating the annular ligament

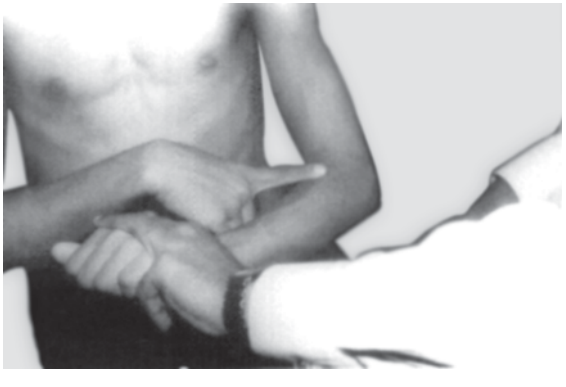


Fig. 6.5: Cozen's test

MOVE

Active movement: Test both arms simultaneously. Ask the patient to touch both shoulders with the hands with arm in abduction to assess active range of full flexion (0 to 135°) or for any limitation. Extension (0 to - 5°) is assessed by similarly asking the arm to stretch out in abduction **(Figs 6.6 and 6.7)**. Forceful passive extension causing posterior elbow pain suggests bony or soft tissue impingement in the olecranon fossa. In throwing atheletes recurrent valgus extension overload cause impingement of proximal medial olecranon leading to osteophyte formation and pain. Supination and pronation is assessed by keeping

Fig. 6.6: Testing elbow extension

both elbows by the side of the body, forearm flexed to 90° and asking the patient to show the palms up for supination (90°) and palms down for pronation (90°) (see **Figs 7.3E and F**).

Passive movement: If the active movements are full there is no need to test passively. In restricted active movements passive movement is done to analyze the range and the nature of restriction, like bony block with definite end point or elastic block with mushy end point from soft tissue tightness.

MEASURE

With elbow flexed 90° assess the distance between 3 bony prominences, medial, lateral epicondyle and tip of olecranon, from the back by holding with the thumb, middle finger and index finger of the examiner respectively. Normally this forms an isosceles triangle and their relationship is altered in elbow dislocation, fractures of epicondyle and condylar fracture of humerus; unaltered in supracondylar fracture **(Fig. 6.8)**. In extension these points form a straight line.

The length of the arm is measured from posterior angle of acromion to lateral epicondyle and forearm is measured from lateral epicondyle to tip of radial styloid.

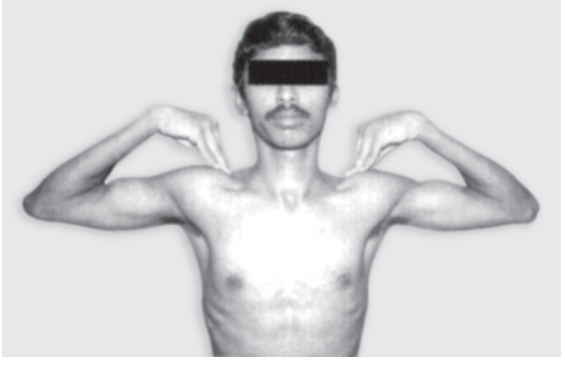


Fig. 6.7: Testing elbow flexion

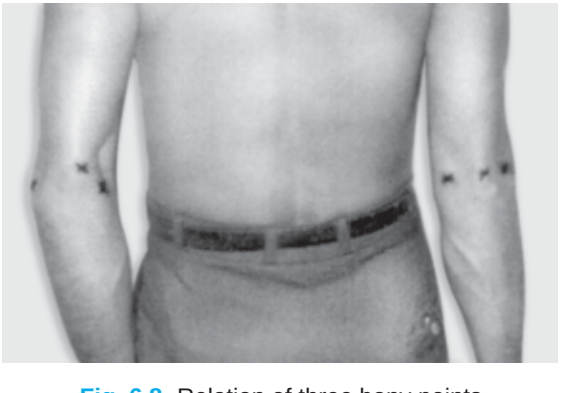


Fig. 6.8: Relation of three bony points

STABILITY TESTS

Medial collateral ligament injury is assessed by valgus stress in supination **(Fig. 6.9)** for abnormal opening up of the joint space. Always compare with opposite elbow.

Lateral collateral ligament injuryis assessed by either varus stress in supination or valgus stress in full pronation **(Fig. 6.10)**.

Patient with extensive lateral ligament complex injury may complain of recurrent

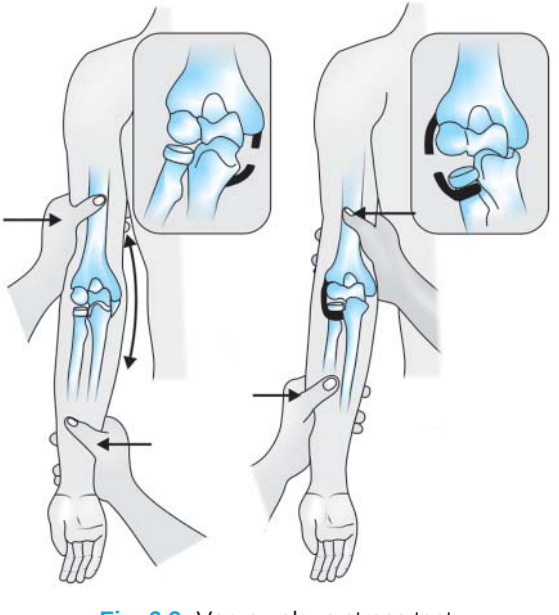


Fig. 6.9: Varus-valgus stress test

painful clicking or locking on extension of the elbow. Posterolateral rotatory subluxation can be present and this is demonstrated by lateral pivot shift test of O'Driscoll where axial compression, valgus stress and supination of the forearm done **(Fig. 6.11)**.

Apprehensive Sign

Asking the patient to rise from a chair using the arms to push them into standing position may reproduce symptoms of instability as it involves axial load, valgus and supination of the forearm. Similar situation occurs in performing press-ups. This will reproduce the patient's symptoms and give rise to apprehension. Subluxation of radius and ulna from humerus causes a prominence posterolaterally and a dimple between radial head and capitellum. When the elbow is at approximately 40° flexion, ulna suddenly reduces with a visible palpable clunk giving rise to apprehension.

Neurological examination consisting of motor strength, reflex testing and sensation testing are done.

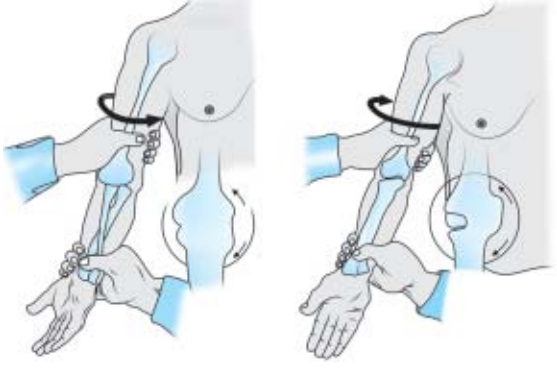


Fig. 6.10: Evaluating the collateral ligaments

Fig. 6.11: Lateral pivot shift test

SPECIFIC CONDITIONS

Supracondylar Fracture

It occurs commonly in children. Pain and swelling at the lower end of humerus is present with limitation of elbow movements. Clinically the three bony points (olecranon, tip of medial and lateral epicondyle) are not altered. Check for distal neurovascular compromise and compartment syndrome. In malunited supracondylar fracture the most common deformity is the gun-stock deformity due to cubitus varus. The distal fragment is in varus, posteriorly tilted and internally rotated causing excessive extension and limitation of flexion. The internal rotation of the shoulder will reveal the amount of excessive internal rotation due to malunion on comparison with the other shoulder. There will be corresponding limitation of external rotation.

Posterior Dislocation of Elbow

This can occur at any age. There will be alteration in the relationship of the three bony points. The triceps may be very prominent and the joint will be swollen due to effusion. Gross restriction of elbow movement with pain will be present. Check for distal circulation and nerve function. It can be associated with elbow fracture.

Pulled Elbow

It occurs in children below 5 years and is produced by traction injury due to lifting or

holding the baby's forearm. The radial head subluxes inferiorly under the annular ligament and the child keeps the arm still and cries with pain. Sudden supination-pronation movement can easily reduce this with a clunk.

Myositis Ossificans (Heterotopic Ossification) (Fig. 6.12)

Elbow joint is notorious for formation of abnormal bony mass in the soft tissue following an injury. This results in stiffness and enlargement of the elbow. Clinically there can be increased warmth with bony thickening and abnormal hard mass in front and back of the elbow. Restriction of elbow movements with hard end-point rather than resilience in soft tissue tightness is felt. It is common in the substance of brachialis muscle.

Olecranon Bursitis (Fig. 6.13)

Swelling of the bursa at the olecranon tip that is cystic, well-localized and translucent. If it is

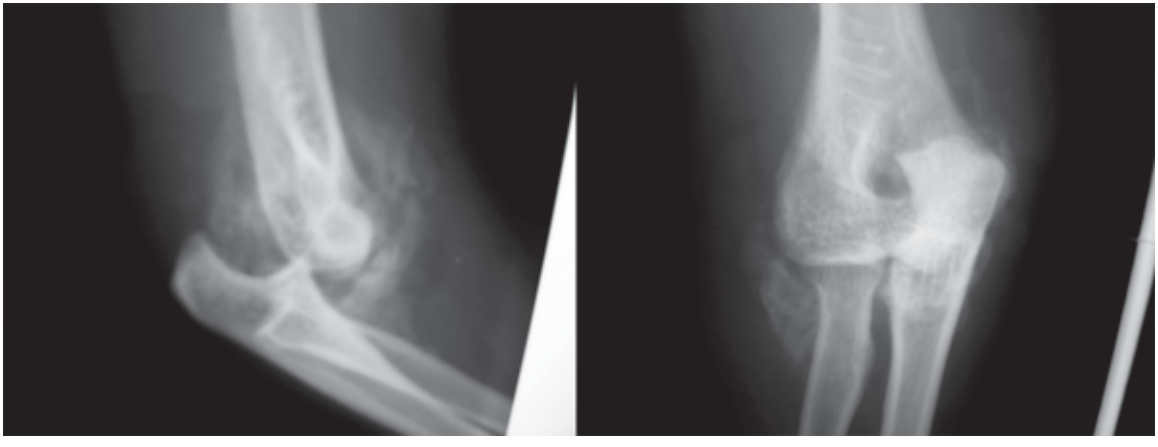


Fig. 6.12: Myositis ossificans in chronic unreduced dislocated elbow

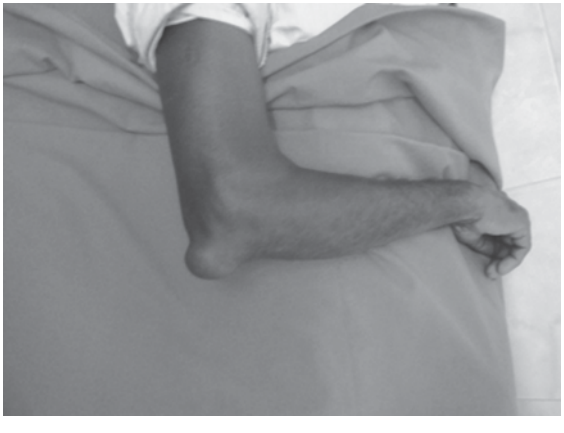


Fig. 6.13: Olecranon bursitis
(For color version, see Plate 2)

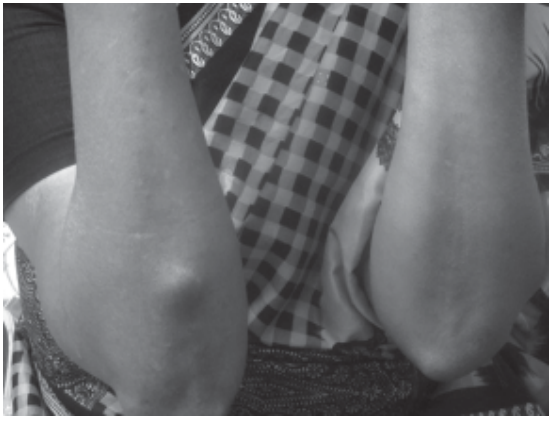


Fig. 6.14: Rheumatoid nodule on extensor aspect of forearm *(For color version, see Plate 2)*

inflamed then signs of inflammation will be present.

Rheumatoid Nodules (Fig. 6.14)

Rheumatoid nodules is present on the extensor aspect of the elbow and forearm, over the subcutaneous border of olecranon. The nodule is firm in consistency and may be multiple.

Congenital Radioulnar Synostosis

Parents complain of child not turning the hand around and always keeps forearm in one position. There will be no supination-pronation movement due to proximal union of radius and ulna. Can have limitation of elbow extension due to developmental defect of radial head. It can be associated with other congenital anomalies such as developmental dislocation of hip and clubfoot.

7 CHAPTER

Examination of Wrist

Wrist is a composite joint formed by radiocarpal joint, ulnocarpal joint, midcarpal joint and distal radioulnar joint. A focused history and well-performed physical examination of the wrist requires knowledge of anatomy and pathology of this area. Based on physical examination, one should be able to make a diagnosis or narrow the differential diagnosis dramatically. This examination is a summation of anatomical locations where symptoms are provoked by palpation and where signs, often with symptoms, are produced by manipulation.

HISTORY

Age, dominant hand and occupation: It is important to know all the three features as it decides the treatment for a problem.

Chief Complaints

Pain

Onset, duration, site, nature, at rest or activity related, aggravating and relieving factors.

Swelling

Onset, duration, site, progression, variation in size, any other swellings, localized or diffuse around the wrist joint, painful or painless and other history as discussed in Chapter 1.

Stiffness

Early morning stiffness, inability to hold things, or perform routine jobs, subjective weakness, loss of wrist movements.

Limitation of Activities

This must be quantified in terms of daily activities and difficulty in performing jobs, pursuing hobbies and leisure activities.

Neurological Symptoms

Weakness, numbness, pins and needles, altered sensation.

Clicks

Snapping sound from carpal instability or tendon subluxation.

Deformity

Dinner fork deformity in Colles' fracture, deformity from malunion or nonunion of distal radius, caput-ulna deformity in rheumatoid arthritis or any congenital deformities, etc. **(Fig. 7.1)**.

Miscellaneous History

Injury, other joint involvement and constitutional symptoms.

Past history, family history, personal history and treatment history must be asked in routine way.

What is patient's expectation?

CLINICAL EXAMINATION

Expose upper limbs, screen neck, shoulder and elbow for any obvious abnormalities or scars. Positioning the patient in sitting and placing the hand on a table is helpful.

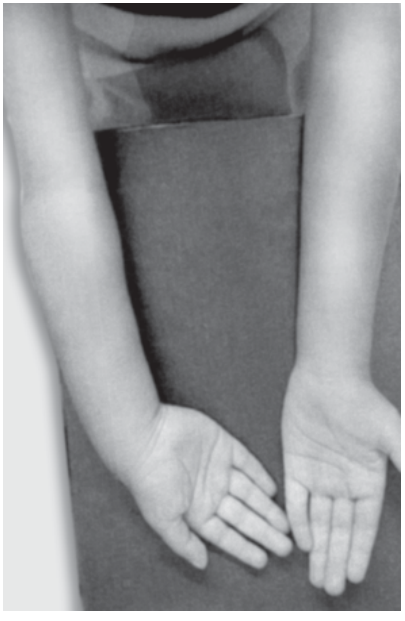


Fig. 7.1: Ulnar club hand

LOOK

From Front, Back and Sides

Attitude of the forearm, wrist and the hand, the shape, swellings, skin color changes, scars, deformities, wasting of small muscles of the hand, pulp atrophy, sweating and nail changes are noted.

FEEL

Ask the patient for most tender spot and examine it last. Look at the patient's face. Define the anatomical spot for tenderness, most of the time this gives the pathoanatomical diagnosis. Swellings are examined as discussed in Chapter 1. Feel the tip of styloid processes of radius and ulna. Normally radial styloid is more distal than the ulnar styloid. Put the nail beds of thumb at 90° to the long axis of forearm, one at radial styloid and other at ulnar styloid. Comparing the respective levels of these land marks give a clinical measure of ulnar variance **(Fig. 7.2)**.

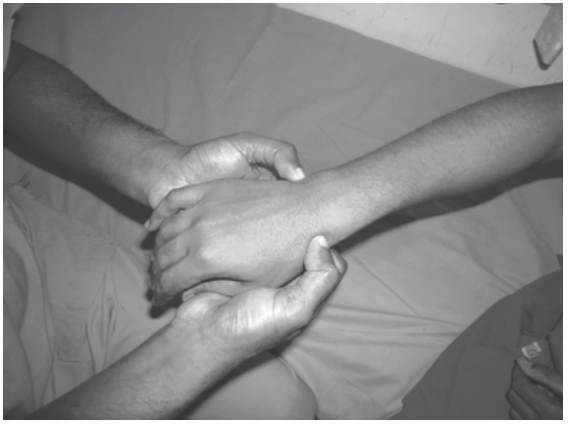


Fig. 7.2: Palpation of radial and ulnar styloid
(For color version, see Plate 2)

MOVE

Active range of following movements are assessed: Dorsiflexion, palmar flexion, ulnar deviation, radial deviation, supination, pronation, full fist formation and full extension. If all movements are present, it is not necessary to check passive movements **(Figs 7.3A to F)**.

Normal range of dorsiflexion is 0 to 70°, palmar flexion 0 to 80°, ulnar deviation 0 to 30° and radial deviation 0 to 20°.

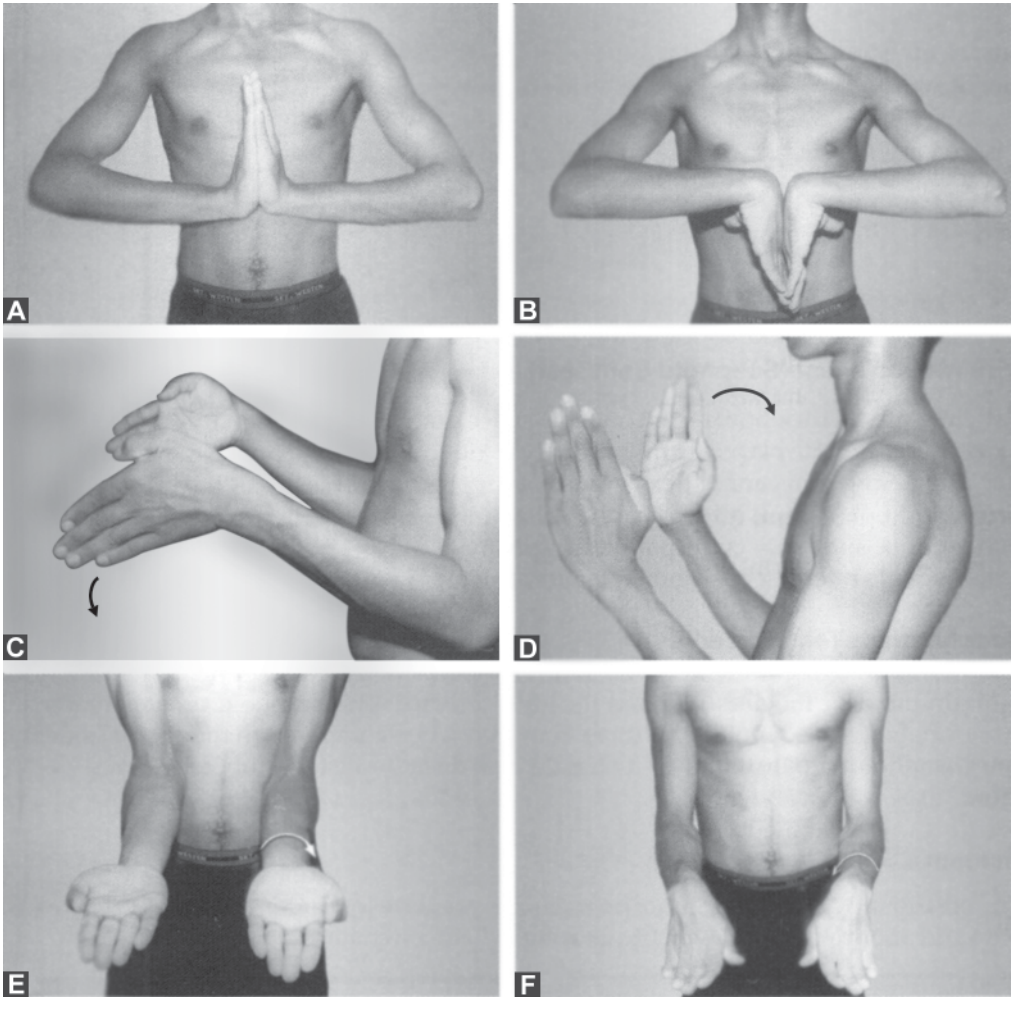
Fixed deformities must be noted and assessed to know whether the deformity is same in all positions of adjacent joints.

ASSESSMENT OF INSTABILITY

Examine the normal wrist first to identify the degree of laxity.

Pseudostability Test (Modified Fisk's Forward Shift Test)

Firmly grip on distal forearm and with other hand grasp the carpometacarpal joints. Patient must be very relaxed and the hand is pressed firmly palmarward. The wrist should be in neutral position. Assess the laxity by comparing with the normal side. Normally there must be palmar translation and if there is any acute pathology in



Figs 7.3A to F: (A) Testing dorsiflexion; (B) Palmar flexion; (C) Ulnar deviation; (D) Radial deviation; (E) Supination and (F) Pronation

the wrist this normal translation will not happen due to spasm of muscles **(Fig. 7.4)**.

Scapholunate (SL) Instability

Kirk Watson's Test (Scaphoid Shift Test)

Scaphoid shift is a provocative maneuver rather than a test, because it does not offer a simple positive or negative result, but rather a variety of findings, with emphasis being on asymmetry on bilateral examination. The maneuver is performed starting with the wrist in slight extension and ulnar deviation. The examiner grasps the wrist from the radial side, placing a thumb on the palmar prominence of the scaphoid while wrapping fingers around the distal radius for counterpressure. The wrist is then passively moved into radial deviation and slight flexion by

Fig. 7.4: Pseudostability test

Fig. 7.5: Kirk Watson's test

the examiner's other hand. The examiner's thumb resists the attempt of the scaphoid tuberosity to rotate volarly, creating a dorsally directed subluxation stress. This subluxation stress causes the proximal pole of the scaphoid to "shift" dorsally in relation to other bones of the carpus and the distal radius even in normal wrists **(Fig. 7.5)**. The degree of the shift is related to the amount of examiner pressure, the degree of scaphoid flexion, the amount of ligamentous laxity, and the status of the scapholunate (SL) ligament. A ruptured SL ligament allows the proximal pole to move more dorsally and frequently rest on the dorsal lip of the radius. The maneuver is best done with the patient's wrist flexed, because this causes the scaphoid to be angled to such a degree that the proximal pole may be only partially constrained by the bony architecture of the dorsal lip of the radius. As the thumb pressure is withdrawn, there may be a palpable "clunk" as the scaphoid returns to its normal position. Pain that replicates the patient's symptoms or asymmetrical laxity when comparing with the contralateral wrist, are considered significant findings. The scaphoid shift maneuver is usually considered a test for SL rupture and scaphoid instability; however, this test is also important for assessing the articular cartilage status of the proximal pole of scaphoid and radial facet, with a gritty sensation or clicking suggesting chondromalacia or loss of articular cartilage. It will also produce symptoms when an occult dorsal ganglion or an occult scaphoid fracture is present. Because the test produces a dorsal displacement of the scaphoid and traction on the SL ligament, if an occult dorsal ganglion is present, the test will generally be painful. Likewise, thumb pressure produces a force that begins on the tuberosity of the scaphoid and travels up the longitudinal axis of the scaphoid. This test will produce a painful stimulus if any fracture exists, and should be considered a mandatory test for all cases diagnosed as "clinical scaphoid fracture".

Scapholunate Test (Shear Test)

One hand of examiner holding the scaphoid, index finger over the scaphoid tuberosity volarly and thumb over the dorsum of the scaphoid area, examiner's other hand holding lunate with thumb over the dorsum press the lunate forwards and scaphoid tuberosity dorsally. Abnormal increased mobility with extreme pain when compared to the normal side indicates scapholunate instability. The lunate is felt distal to Lister's tubercle in mid-dorsum **(Fig. 7.6)**.

Lunotriquetral (LT) Instability

The LT Compression Test

Load the LT joint in an ulnar-to-radial direction, eliciting pain with LT instability or degenerative

Fig. 7.6: Scapholunate ballottement test

Fig. 7.7: Triquetrolunate ballottement test

joint disease. The examiner's thumb applies a radially direct pressure on the triquetrum just distal to the ulnar styloid at the "ulnar snuffbox," the space between the tendons of flexor carpi ulnaris and extensor carpi ulnaris. This maneuver is similar to the radiocarpal glide test described for radiocarpal instability **(Fig. 7.8)**.

Reagan's Shear Test

Thumb dorsally on lunate, index finger applies pressure to triquetrum volarly. This causes pain and abnormal mobility in triquetrolunate ligament injury.

Masquelet's Ballottement Test

Examiner uses both hands to apply shear force across articulation. Examiner's index finger placed on the pisiform bone volarside, which lies on the surface of the triquetrum, the other hand holding the lunate with the thumb on the dorsum, triquetrolunate joint is stressed anteroposteriorly. The shear test is assessed for abnormal mobility and pain, and compared with the normal side. This indicates triquetrolunate instability. The landmark for the triquetrum is the pisiform bone, which is the most prominent bony landmark on volar and ulnar aspect of the wrist **(Fig. 7.7)**.

Radiocarpal Instability

The anteroposterior drawer test can be used to assess for instability of either the radiocarpal or midcarpal joints. The examiner stabilizes distal forearm with one hand while the other hand grips the metacarpals, applying longitudinal traction and an anteroposterior force. A "drawer" is elicited though the radiocarpal or midcarpal joint, and compared with the contralateral side.

The radiocarpal glide will test the articular surface of the proximal carpal row and the extrinsic ligaments **(Fig. 7.8)**. The examiner's thumb exerts a radially directed force on the triquetrum. A radial shift of the proximal carpal row relative to the distal radius may be appreciated in the setting of radiocarpal instability. Crepitus may be felt in the setting of articular pathology of the radiocarpal joint.

Midcarpal Instability

Midcarpal Shift Test of Lichtman

Normally as the wrist moves from radial to ulnar deviation, the proximal carpal row rotates smoothly from flexion to extension while the distal row translates from palmar to dorsal. With midcarpal instability, the proximal row remains flexed and the distal row remains volarly

translated longer than normal during ulnar deviation. As ulnar deviation progresses, the soft-tissue and bony restraints cause a sudden "catch-up" of the proximal row into extension and the distal row into dorsal translation, which is often an audible or palpable "clunk". With the wrist in neutral ulnoradial deviation the examiner stabilizes the forearm in pronation with one hand, and with the other hand applies a palmarly directed pressure at the level of the distal capitate, noting the ease and extent of palmar translation. The wrist is then axially loaded and passively ulnar deviated. The test is positive if a painful catch-up clunk occurs with ulnar deviation that reproduces the patient's symptoms. The presences of palmar translation or a clunk alone without the reproduced symptoms are not considered positive, because they can occur in normal asymptomatic patients **(Figs 7.9A and B)**.

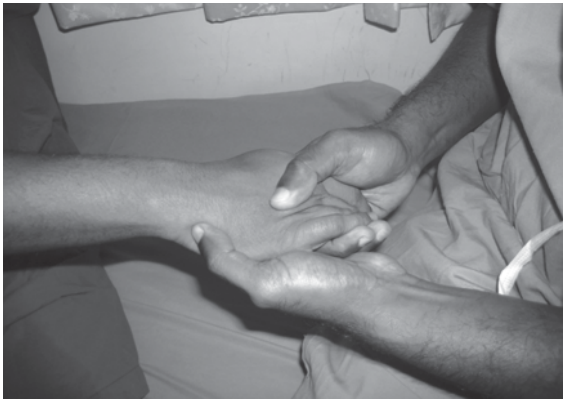
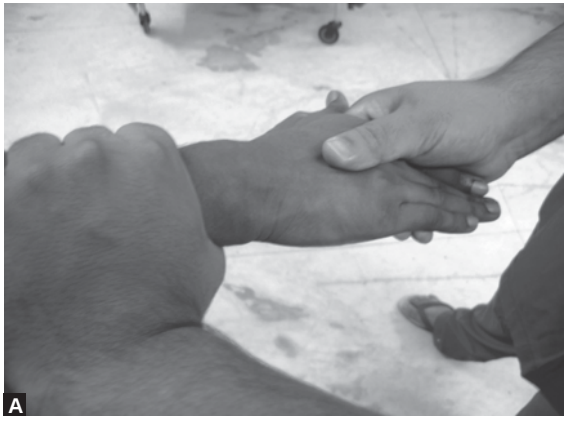


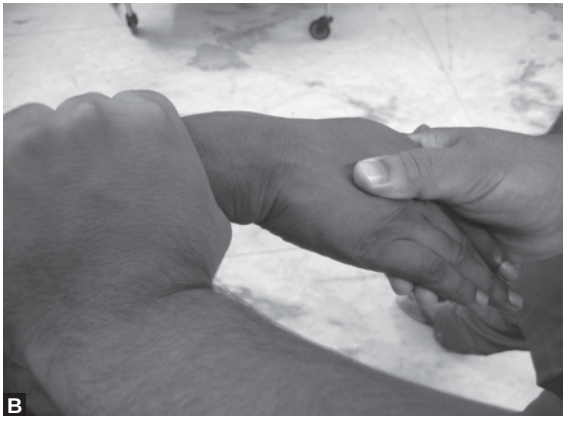
Fig. 7.8: Radiocarpal glide test *(For color version, see Plate 2)*

ASSESSMENT OF RADIAL WRIST PAIN

Pain can be due to scaphoid fracture, non union, styloid fracture, SLAC (scapholunate advanced collapse) lesion, scapholunate instability, radiocarpal arthritis, scapho-trapezio-trapezoid arthritis (Triscaphe), trapeziometacarpal arthritis, tendonitis—de Quervain's tenosynovitis and flexor carpi radialis tendonitis. Superficial branch radial neuritis—Wartenberg's chieralgia characterized by pain and tenderness 1 to 2 cm proximal to the radial styloid, and radicular pain distally along the course of the superficial radial nerve elicited by percussion. Pain in this structure is much more likely related to a traumatic neuroma, peritendinitis crepitans (intersection syndrome).

Palpate the distal palmar tuberosity of the scaphoid. Curl one's fingers about the radial aspect to the dorsum of the patient's wrist while the thumb is palmar and points distally **(Fig. 7.10)**. This is located immediately proximal to the thenar





Figs 7.9A and B: Midcarpal shift test *(For color version, see Plate 3)*

eminence and immediately radial to the flexor carpi radialis tendon. Use the opposite hand to move the patient's hand/wrist unit into flexion-extension and radioulnar deviation. If one is palpating the distal pole of the scaphoid, this small bony lump will move, demonstrating that it is part of the carpus and not the radius. More importantly, the distal pole will become prominent palmarly with wrist flexion and with radial deviation as the scaphoid rotates into flexion.

Adjacent and immediately ulnar to the scaphoid tuberosity is the tendon of the flexor carpi radialis (FCR). This can often be visualized proximally, and if' not, it can be palpated. Follow it proximally by laying three fingers on it while palmar and dorsiflexing a clenched fist. FCR tendonitis can manifest as tenderness upon palpation distally near the fibro-osseous tunnel in the trapezium as it dives to insert into the base of the second metacarpal. There is usually localized pain with hyperextension of the wrist caused by tendon stretch and with resisted wrist flexion and radial deviation.

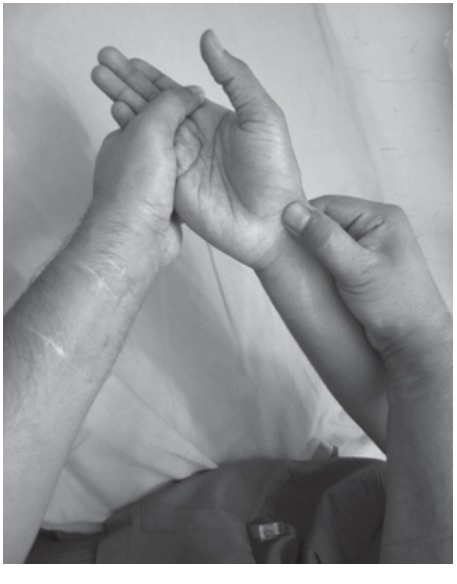


Fig. 7.10: Palpation of distal tuberosity of scaphoid
(For color version, see Plate 3)

Immediately radial to this point and distal to the scaphoid tuberosity is the scaphotrapezial (ST) joint. At this location, place your thumb nail transversely and at 90° to the long axis of the forearm. Ask the patient to move his thumb. There will be an appreciation of movement distally while the scaphoid tuberosity remains still. This will be useful for localizing pain related to ST arthritis, a common cause of radial palmar wrist pain, and to localize the entry point for an injection into that joint.

Finkelstein's Test

De Quervain's tenosynovitis is assessed by thumb in palm and ulnar deviation of the wrist. This causes pain over the radial styloid region and along the tendon sheath. The first dorsal compartment, which has abductor pollicis longus and extensor pollicis brevis gets inflamed **(Fig. 7.11)**.

Intersection syndrome, also known as peritendonitis crepitans, is an overuse condition resulting in inflammation in the area where the muscle bellies of the APL and EPB cross the underlying extensor carpi radialis longus (ECRL) and brevis (ECRB) tendons. The underlying pathologic abnormalities include stenosing tenosynovitis of the tendon sheath of ECRL and ECRB tendons or APL bursitis. It presents as pain, swelling, tenderness, and crepitus in the radiodorsal forearm about 4 cm proximal to the

Fig. 7.11: Finkelstein test

tip of the radial styloid, corresponding to the intersection of the first and second extensor compartments. Finklestein's test is often painful with APL bursitis, although the pain is usually more proximal in the radiodorsal forearm.

Grinding Test

Axial loading of the first metacarpal and twisting around causes severe pain and crepitus in trapeziometacarpal arthritis **(Fig. 7.12)**. Usually this is accompanied by palpable crepitus and a painful sensation. If the subluxation is more than 2 or 3 mm, the outline of the thumb will form a slight step, called the "shoulder sign".

Scaphoid Pathology

Tenderness in anatomical snuffbox and axial loading of first metacarpal can cause pain at dorsoradial aspect of wrist. Examiner's index finger palpating just distal to radial styloid with wrist moved passively from radial to ulnar deviation can palpate articular/non-articular junction of scaphoid. It is painful in scaphoid nonunion, periscaphoid synovitis, scaphoid instability or SLAC changes at styloid.

The Snuffbox

Distally in the snuffbox, the palmar border is formed by the first dorsal compartment tendons.

Fig. 7.12: Grinding test

The dorsal border is formed by the combined second and third compartments. The proximal border is the distal radius and the distal border the base of the first and second metacarpals. Spend a moment and find these limits. The snuffbox contains fat, the radial artery traversing obliquely, and the wrist joint capsule. Through this capsule the waist of the scaphoid can be readily felt when the wrist is ulnarly deviated. The junctional point along the radial border of the scaphoid, where the proximal articular surface changes to nonarticular surface, is referred to as the scaphoid articular-nonarticular (ANA) junction. With the wrist in ulnar deviation, the ANA junction can be palpated with the examiner's index finger placed just distal to the radial styloid. Whereas mild tenderness is present there in normal wrists, scaphoid instability or synovitis is said to result in more severe pain. Asymmetry on bilateral examination is important.

Move to the dorsal border of the snuffbox and realize that this border consists of both superficial and deep components. The extensor pollicis longus (EPL) forms the superficial border and heads toward the thumb. Deep to this is the ECRL tendon. Extend the interphalangeal (IP) joint of the thumb and feel the EPL. Dorsiflex the wrist and feel the ECRL. Follow the ECRL distally to its insertion in bone. Make a clenched fist and put the tip of the index finger into the V that forms distally between the ECRL and ECRB. Extend the IP joint and abduct the thumb. The EPL should stand out visibly and be easily palpable through its course to the mid-dorsal radius, where it courses about the ulnar aspect of Lister's tubercle. Feel this definite short oblong bump with the tendon moving next to it. Feel the beginning of the radiocarpal joint just 2 to 3 mm distal to this tubercle. Move the wrist into dorsi and palmar flexion and be certain that the "lump" remains stationary.

Next, hold the hand and apply thumb pressure in the interval between the two arms of the V made by the ECRL and ECRB. Flex and extend the wrist. In flexion, appreciate a smooth firm bump becoming prominent in this interval. This is the dorsal proximal

pole of the scaphoid covered by capsule. It should be firm and not painful to press on. Scaphoid impaction is a condition in which repetitive hyperextension of the wrist causes impingement of the scaphoid onto the dorsal lip of the radius. A tender dorsal osteophyte or spur on the dorsal radial lip or dorsal scaphoid rim may be palpable, and extension of the wrist may be limited or painful.

STT (scapho-trapezio-trapezoid) joint pathology: This joint is felt by following the course of 2nd metacarpal proximally with examiner's thumb until it falls into a recess. It is painful in STT synovitis, degenerative disease or other scaphoid pathology.

Scapholunate joint pathology: Follow the course of 3rd metacarpal proximally until the examiner's thumb falls into a recess over capitate. Scapholunate joint is just proximal between extensor carpi radialis brevis and 4th dorsal compartment. It is tender in Keinbock's disease and scapholunate dissociation.

ASSESSMENT OF ULNAR WRIST PAIN

The conditions that may cause ulnar wrist pain are triangular fibrocartilage complex injuries (TFCC) more common in radial fracture malunion, tendonitis, ulna-carpal abutment syndrome or impaction syndrome, pisotriquetral arthritis, triquetrolunate instability (VISI), hamate fracture, extensor carpi ulnaris subluxation and caput-ulna in rheumatoid arthritis.

Triangular fibrocartilage complex (TFCC) consists of articular disk, meniscus homologue, ulnar carpal ligament, dorsal and volar radioulnar ligaments, and extensor carpi ulnaris sheath. It is important in loading and stabilizing the distal radioulnar joint. It can get torn due to degeneration or trauma. This is assessed by elbow resting on the table, holding the hand in 'shake-hand position', the other hand supporting the forearm apply axial load in ulnar deviation of the hand and do supination-pronation movement. This produces extreme pain on the ulnar aspect

Fig. 7.13: TFCC test

of wrist **(Fig. 7.13)**. This "grind test" will be positive in ulnocarpal impaction or TFCC tear.

With the forearm pronated, palpate ulnar and distal to the ulnar head. Deviate the wrist radial and ulnar, and feel the tendon of the extensor carpi ulnaris (ECU) become prominent on ulnar deviation. Trace this tendon distally to its insertion into the dorsoulnar base of the fifth metacarpal. Tenderness along the tendon sheath indicates tendonitis.

The ulnar styloid (US) is best felt when the forearm is pronated. It is distal to the ulnar head and palmar to the ECU. It is slightly obscured by the ECU when the forearm is supinated. It should not be tender to palpate unless there has been a recent fracture or ulnar styloid-triquetral impaction (USTI) is present. To search for clinical support for this diagnosis, a USTI provocative test is performed. This USTI test is based on the fact that the US is ulnar in pronation, and is more central and dorsal in supination. Thus it is evident that to approximate the US to the triquetrum, one needs to bring the US closer to the carpus by supinating the forearm, and bring the carpus closer the wrist dorsiflexed and the forearm pronated, and simply add one motion, supination, while maintaining dorsiflexion **(Fig. 7.14)**. To support the diagnosis the US should also be tender exactly over its tip. This is tested in pronation and neutral wrist flexion. The patient may indicate from the history that this test produces pain. The

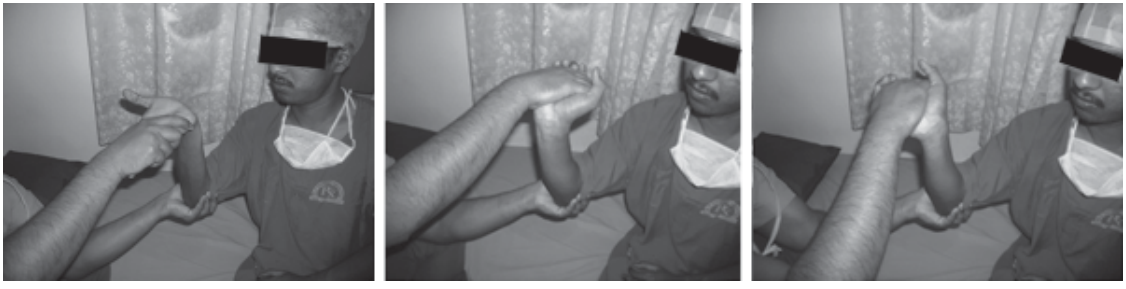


Fig. 7.14: USTI provocative test *(For color version, see Plate 3)*

pain with the hand in the back pocket, repetitive page turning, or the distal supinated hand on the ice hockey stick may be historical evidence of a positive USTI provocative test.

The lunotriquetral (LT) joint can be localized, it is a depression just distal to the radial side of the ulnar styloid, because the head of the ulna articulates with one half of the lunate and one half of the triquetrum. Direct palpation of the LT joint may be tender when LT pathology is present.

ASSESSMENT OF DORSAL WRIST PAIN

Scapholunate Interval

Move ulnarly and place your thumb just distal to the dorsal lip of radius in line with the long metacarpal. Flex and extend the wrist and feel a poorly defined hard lump becoming prominent in flexion. This is the dorsal pole of lunate. It is covered by capsule, extensor digitorum longus, tenosynovium, and retinaculum, and is not felt very distinctly-but it is felt. Pressure on this area is generally not painful unless a fracture or Kienbock's disease is present. Appreciate the hard fullness felt with palmar flexion, and move back and forth between the dorsal pole of the lunate and proximal pole of the scaphoid. Palpate the intervening SL area. Appreciate the slight valley that exists. This area should not be painful unless there is a recent SL ligament tear or a chronic occult ganglion. This is usually the area where the dorsal ganglion becomes obvious.

Fourth and Fifth Extensor Compartment

The extensor digitorum communis (EDC) tendons (fourth compartment) and their tenosynovium is easily appreciated by flexing and extending the fingers at the MCP joints. This can be done as a unit, but is better appreciated if done in rhythmical consecutive fashion. Similarly, place the fingers in a "piccolo" fashion longitudinally between the EDC and head of ulna, and flex and extend the little digit. The tendon of the extensor digiti minimi (EDM) can be felt moving. Tenosynovitis is a common source of pain, swelling, and tenderness in the dorsuin of the wrist. Ganglion cysts and vestigial wrist extensor muscles (extensor digitorum brevis minus) are less common but may have a similar presentation.

Carpometacarpal Joints

Sprains of the second through fifth CMC joints can be associated with localized tenderness and swelling. Stressing the joint by flexion, extension, and rotational forces may add additional information. A bony prominence at the base of second or third metacarpal, often involving the CMC joints, is called a carpal boss. The cause and significance of this prominence is unknown, and caution is suggested when considering any surgical treatment.

Ganglion: It is a cystic, well-localized swelling with positive transillumination test. Dorsal ganglion results from cystic myxomatous degeneration

within the dorsal scapholunate ligament and may be related to scapholunate instability.

Keinbock's disease: It is avascular necrosis of the lunate and is associated with ulna minus variant. Clinically patient will have tenderness on mid-dorsum over the lunate bone.

ASSESSMENT OF PALMAR WRIST PAIN

In the palmar ulnar aspect hold the pisiform between the index finger and thumb. Flex and extend the wrist and move the pisiform medially and laterally while applying dorsally directed pressure, compressing the pisiform on the triquetrum, to search for articular cartilage crepitus or pain associated with pisotriquetral degenerative joint disease. This is referred to as the pisotriquetral grind test **(Fig. 7.15)**.

Palpate the hook of the hamate just distal and radial from the pisiform. It is localized by placing the IP joint of the examiner's thumb over the more superficial pisiform, with the tip of the thumb directed toward the metacarpal head of the long finger. Deep palpation with the tip of the examiner's thumb reveals the hook of the hamate. This can be tender in the setting of fracture or nonunion of the hook of the hamate. Remember that this is the area of the ulnar nerve, and deep palpation onto this nerve is usually painful.

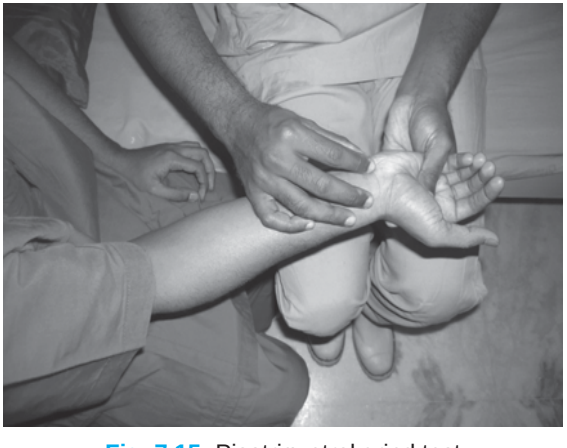


Fig. 7.15: Pisotriquetral grind test
(For color version, see Plate 4)

Palpate the flexor carpi ulnaris (FCU) proximally from the pisiform. It is most prominent by having the patient make a clenched fist during mild wrist flexion. Tenderness along the tendon sheath or pain and weakness with resisted wrist flexion and ulnar deviation suggest tendonitis.

With the tip of the thumb on the radial palmar side of the pisiform, add deep pressure. The uncomfortable sensation is related to pressure on the ulnar nerve. Although one cannot objectively feel this nerve, this means of localization will be of value for assessing symptoms or injecting local anesthetic.

The palmaris longus (PL) tendon is central and superficial in the palmar distal forearm. It stands out with a flexed grip, and can be visualized and palpated. It may be absent. At the wrist crease between the PL and FCR, an astute examiner can often palpate it fine snapping of the palmar cutaneous branch of the median nerve. This subtle finding is aided by tensioning the nerve with dorsiflexion of the wrist and then drawing the tip of the examining digit across the interval with slight deep pressure. Finally, circumferential wrist compression with the thumb and index will produce pain when a synovitis and effusion is present.

Palmar wrist pain can be from palmar ganglion that arises from scaphotrapezial ligament or a compound palmar ganglion from radial bursitis in rheumatoid or tuberculous synovitis (presence of cross-fluctuation proximal and distal to flexor retinaculum). Rarely it can be referred pain.

ASSESSMENT OF DISTAL RADIOULNAR JOINT (DRUJ)

Tenderness over distal radioulnar joint with subluxation of the ulnar head dorsally indicates DRUJ pathology.

Shear Test or Distal Ulna Ballottement Test (Fig. 7.16)

Holding the distal end of radius and ulna with thumb and fingers of each hand individually elicit shearing movement, this causes pain in

Fig. 7.16: DRUJ test

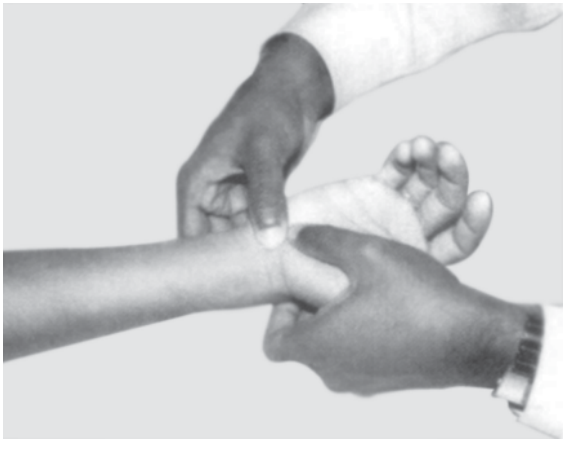


Fig. 7.17: Carpal compression test

degenerative changes, chondromalacia or osteochondral injuries.

Ulnar Compression Test

It can also be confirmed by pressing ulnar head against the sigmoid notch by spring test of distal radius and ulna or test supination/pronation with DRUJ squeezed together.

Piano Key Test

The examiner depresses the ulnar head palmarly while the pisiform is stabilized with a dorsally directed force. A positive piano key sign is when the ulnar head springs back into position like a piano key when the forces are released. Pressing the dorsum of the ulna with one finger elicits pain at DRUJ.

Differential Lignocaine Injection Test

Differential Lignocaine Injections can be Incorporated into the Physical Examination to Help Localize the Source of Pain

SPECIFIC CONDITIONS

Carpal Tunnel Syndrome

1. Tinel's sign may be elicited over median nerve.
2. Direct carpal compression test is most diagnostic which elicits pain and paresthesia along the median nerve distribution. This test is more sensitive than Phalen's test **(Fig. 7.17)**.
3. Phalen's test keeping the wrist flexed for 30 seconds elicits pain and paresthesia along the median nerve distribution **(Fig. 7.18)**.
4. Wasting of thenar muscles and hypoesthesia on radial three and half fingers of the hand can be present in long-standing cases.
5. Strength of abductor pollicis brevis is assessed by keeping the hand flat with the palm facing up and asking the patient to touch the tip of the examiner's finger with the thumb in the plane of abduction and feel for muscle.
6. Opponens pollicis is tested by making the thumb tip opposed to little finger and assessing the ability to keep opposed against resistance.
7. The sensation over thenar eminence will be normal as it is supplied by palmar cutaneous branch of median nerve.

Colles' Fracture

Fracture of the distal radius at corticocancellous junction (2 cm from the articular surface) characterized by dorsal displacement, dorsal tilt, lateral displacement, lateral tilt, impaction and supination of distal fragment.

Clinically it manifests with pain, swelling, bruise, limitation of movements and dinner-fork deformity. It is a common fracture in the elderly population from fall on outstretched hand due to osteoporosis. Complications include Sudeck's atrophy, extensor pollicis longus rupture, malunion, nonunion and post-traumatic stiffness.

Smith Fracture

Fracture of distal radius at metaphysis with volar displacement or tilt of the fragment.

Barton's Fracture

Intra-articular fracture of the distal radius with displacement of the carpus along with the distal intra-articular fragment. It can be of volar or dorsal displacement.

Malunited distal radius ***(Fig. 7.19)***: Malunited distal radius manifests with deformity and thickening of the radius. The levels of styloid of the radius and ulna are assessed. Both styloids are at same level or radial styloid is more proximal than ulna due to shortening of radius. Distal radioulnar joint is assessed as described earlier. Movements are limited depending on the tilt of the distal fragment and intra-articular extension. For example, a dorsally tilted articular surface of the distal radius limits palmar flexion and may have excessive dorsiflexion as the arc of movement has changed.

Tendonitis

In tendonitis or tenosynovitis there is oblong or longitudinal swelling along the course of the tendon, stretching the tendon passively can be painful and soft crepitus can be elicited on flexion and extension movements, e.g. extensor carpi ulnaris or flexor carpi ulnaris tendonitis.

Gamekeeper's Thumb or Skier's Thumb (Fig. 7.20)

Ulnar collateral ligament injury of thumb that usually tears at its distal attachment to the proximal

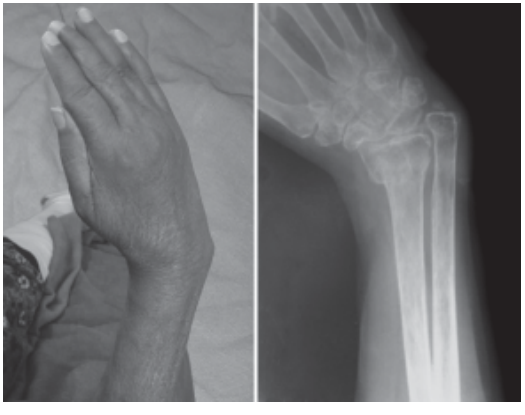


Fig. 7.19: Malunited Colles' fracture with X-ray showing gross radius shortening, radial deviation and flexion deformity of the wrist

Fig. 7.18: Phalen's test

Fig. 7.20: Gamekeeper's thumb

phalanx. The ligament can become displaced outside of the adductor aponeurosis (Stener's lesion) and cannot heal without operative intervention. Avulsion fracture is treated according to the displacement. Clinically this injury can be identified by pain, swelling and tenderness at ulnar aspect of base of the thumb. On valgus stress test in 30° flexion of MCP joint the joint opens up. This leads on to chronic instability compromising pinch grip when the thumb is stressed in abduction and hence functional disability. Compare with opposite thumb for abnormal movement on valgus stress. In acute painful situation infiltration of local anesthesia and valgus stress confirming with stress X-ray may be of good help.

Madelung's Deformity

Manus varus and flexus deformity of the wrist due to developmental abnormality of distal radius physis. The volar and ulnar aspect of epiphyseal plate growth is disturbed resulting in this deformity. This can be from dysplasia (developmental), trauma, infection and metabolic causes. The developmental type is more common in females, usually bilateral and can be asymptomatic. The patient can present with wrist pain, deformity or weak grip strength. Clinically there is dorsal bayonet like deformity of radius with shortening and dorsal prominence of head of ulna which is relatively long. Limitation of dorsiflexion and increased flexion of the wrist can be demonstrated. TFCC tears are common and can cause pain, and this can be confirmed with stress test.

Radial Club Hand

Radial agenesis may be total or partial longitudinal deficiency. Radial deviation of wrist due to absent or partially developed radius and hand with absence of radial carpus or thumb ray can be present.

It can be associated with Holt-Oram syndrome, TAR (thrombocytopenia absent radius) or VATER (Vertebral anomalies, anal anomalies, tracheoesophageal fistula, and renal anomalies) syndromes. Elbow mobility decides the management. Good elbow movements is a prerequisite for centralization procedures to ensure hand to mouth feeding.

Carpal Boss

Benign bony hard swelling at the 2nd or 3rd carpometacarpal joint dorsally.

Ganglion

Myxomatous degeneration of capsule or tendon sheath resulting in tense globular swelling usually in the mid dorsum of wrist (from scapholunate dorsal ligament). Cystic swelling with positive cross fluctuation test or Paget's test. Volar ganglion is common over radial aspect from radioscaphoid joint.

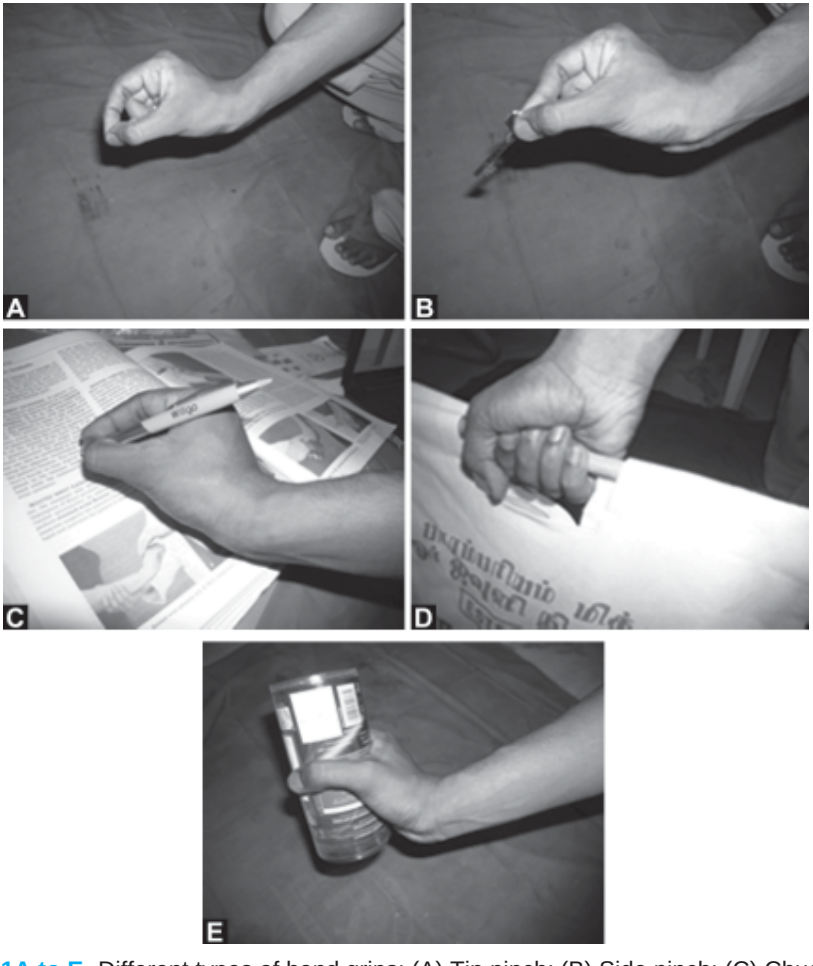
Wrist Synovitis

Diffuse swelling and tenderness all round the joint can be from rheumatoid synovitis, tuberculosis and rarely secondary osteoarthritis.

8 CHAPTER

Examination of Hand

The primary function of the hand is sensation and grasping. The dexterity and functions like grasp, side pinch (key pinch), tip pinch, chuck pinch, hook and fist are important for day-to-day activities **(Figs 1A to E)**.



Figs 8.1A to E: Different types of hand grips: (A) Tip pinch; (B) Side pinch; (C) Chuck pinch; (D) Hook grasp, and (E) Power grasp *(For color version, see Plate 4)*

As in any upper limb problem the history should include:

a. Dominant hand
b. Occupation
c. Functional impairment as seen in job, daily activities of eating, dressing, perineal hygiene, fastening button, turning a tap, using a key, holding a cup, opening jars, etc.
d. Involvement of other joints.

Patient can complain of pain, swelling, stiffness, deformity, paresthesia, numbness, weakness of hand and patient's expectation must be known. In acute trauma, always document the time, site, and description of the accident. Record the type of injury as cuts, crush injuries, saw accidents, chemical or electric burns, bite wounds and closed trauma. A thorough history will provide sufficient information for a tentative diagnosis. For example, altered sensation and weakness in the index and middle finger accompanied by night time paresthesia is typical of carpal tunnel syndrome. Sudden painful snapping over the metacarpal heads when flexing and extending the finger is typical of trigger finger.

Expose whole of both upper limbs. Screen the neck, shoulder and elbows especially in rheumatoid arthritis. Ask the patient to do neck movements, lift both hands above shoulder, flex and extend elbows, supinate and pronate forearm, and palmarflex and dorsiflex wrist. This gives a useful quick screening of the above joints.

LOOK

From proximal to distal and in pronation and supination of the hand. Comment on the skin condition, color—localized hyperemia and erythema in infection of hand, hyperpigmentation of palmar furrows in Addison disease, shiny atrophic skin in progressive scleroderma, attitude, swellings, scars, muscle wasting, pulp and nail changes, vasculitis, deformities, rheumatoid nodules, Heberden's nodes (degenerative joint disease of DIPJ) and Bouchard's nodes (degenerative joint disease of PIPJ). Dry scaly skin is a sign of loss of nerve function because of inability to perspire. Nail changes can be pitting from psoriasis, medical clubbing, splinter hemorrhages indicating vasculitis and sometimes extremely painful subungual glomus tumors appearing as spot of purplish blue under the nail.

Look for the normal attitude of the hand. Any deviation may be due to tendon injury or rotation from fractures **(Fig. 8.2)**.

Loss of skin creases can be from swelling or post-traumatic sympathetic dystrophy or arthrogryposis multiplex congenita.

Deformities are zig-zag pattern in inflammatory joint disease. They can be:

1. Dorsal subluxation of ulna at distal radioulnar joint **(Fig. 8.3)**.
2. Palmar subluxation of the radiocarpal joint.
3. Radial deviation of metacarpals.
4. Ulnar deviation of fingers **(Fig. 8.4)**.
5. Boutonnière deformity of fingers (PIPJ flexion and DIPJ extension) **(Fig. 8.5)**.
6. Swan neck deformity (PIPJ extension, DIPJ flexion and sometimes MCPJ flexion) **(Fig. 8.5)**.
7. Thumb deformities: MCPJ flexed, IPJ extended; MCPJ extended and IPJ flexed.
8. Z deformity CMCJ adducted and subluxed.
9. Dropped fingers: Inability to extend little or ring finger (Vaughen-Jackson syndrome) due to extensor tendon rupture from dorsal subluxation of ulna.

Fig. 8.2: Normal attitude of hand: Increasing flexion attitude of ulnar fingers *(For color version, see Plate 5)*

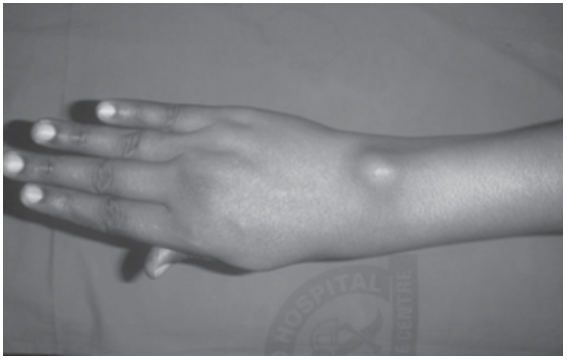


Fig. 8.3: Dorsal subluxation of ulna at distal radioulnar joint *(For color version, see Plate 5)*

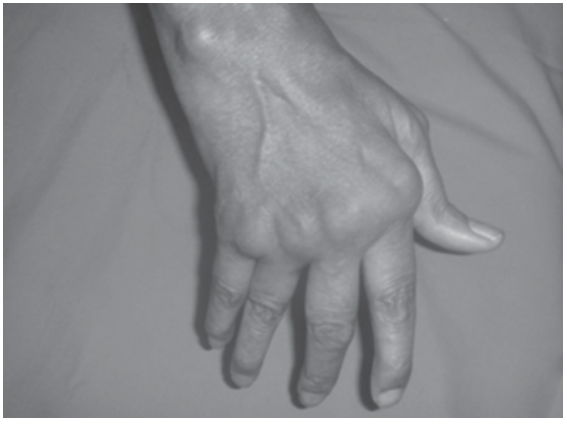


Fig. 8.4: Ulnar deviation of fingers *(For color version, see Plate 5)*

10. Mallet finger: DIPJ is flexed and patient is unable to actively extend tip of the finger, but passive movement is possible. This is due to rupture of extensor tendon resulting from trauma, inflammation, degeneration or avulsion fracture.

FEEL

a. Tenderness—define the exact anatomical area.
b. Swelling—examine in usual manner.
c. Synovial thickening—Pinch the skin on the volar aspect of proximal phalanx, inability to pinch indicates tenosynovitis (Bovier's sign).

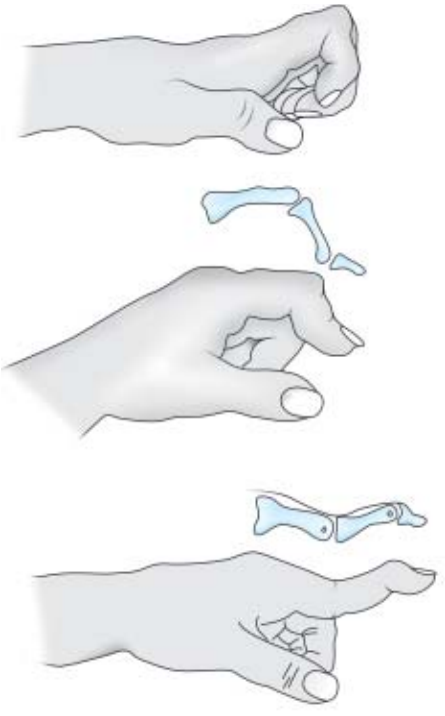


Fig. 8.5: Claw hand, boutonnière and swan neck deformity of index finger

d. Check by passive movement, the fixed deformity or lag. If there is a deformity, check whether the deformity is same in all positions of adjacent joints.
e. Fine crepitus in synovitis and snapping sound in trigger finger.
f. Grip strength and fine pinch—This can be assessed using a dynamometer or simply by asking the patient to squeeze the examiner's finger in his hand.
   Check pinch grip by holding a key or coin.
g. Neurological examination.

MOVE

Check the active range of movements and pain on movements and then passive movements.

At wrist check for active supination, pronation, palmar-flexion, dorsiflexion, radial and ulnar deviation.

At fingers check for active flexion, extension, abduction and adduction.

MCPJ moves from 0 to 90°; PIPJ from 0 to 110°; and DIPJ from 0 to 90°.

Check individual flexor tendon and extensor tendon function for tendon injury or nerve injury. Differences in active and passive ranges of motion according to the position of the joint, narrow the differential diagnosis. For example, equal limitations in the active and passive ranges of motion of a joint, regardless of the position of the joint, are a sign of shortening of capsule and ligaments or intra-articular incongruity. An extension deficit in a PIPJ that does not change as MCPJ moves is a sign of adhesion of the flexor tendon at the level of MCPJ. Adhesion of the extensor tendon in the forearm prevents flexion in the wrist when the patient makes a fist; adhesions of the flexor tendons prevent extension with fingers extended. Impairment of active movement of only one finger is a sign of a tendon rupture or a neurological lesion.

At thumb check for flexion, extension, adduction, abduction and circumduction.

Joint stability is tested by stressing the collateral ligaments, for example in game keeper's thumb radial deviation of thumb is done to assess ulnar collateral ligament of MCPJ.

SPECIFIC CONDITIONS

Claw Hand or Intrinsic Minus Hand (Fig. 8.6)

The deformity is hyperextension of MCPJ and flexion of PIPJ. This results from intrinsic muscles (interossei and lumbricals) paralysis in ulnar nerve palsy (little and ring finger), median nerve palsy (middle and index finger), Volkmann's ischemic contracture of hand and Charcot-Marie-Tooth disease.

Differential Diagnosis

1. Post-traumatic contracture
2. Dupuytren's contracture
3. Camptodactyly

Fig. 8.6: Claw hand

Intrinsic Plus or Tightness of Hand

The deformity is flexion of MCPJ and extension of IPJ. This results from tightness or contracture of the intrinsics due to ischemia or inflammation as in rheumatoid arthritis. Bunnel's test is done to identify the tightness. PIPJ flexion movement is limited with MCPJ in extension and becomes more with MCPJ in flexion. In rheumatoid patient, reduce the MCPJ subluxation and correct ulnar deviation before testing for intrinsic tightness.

Quadrigia Effect

It presents with loss of maximum active flexion and decreased grip strength in adjacent digits. This occurs in flexor digitorum profundus (FDP) lacerations distal to lumbrical origin resulting in tight lumbricals than extrinsics, or digital amputation where flexors are sutured to extensors or in tenodesis of FDP or poor flexor tendon repairs. This occurs due to mass action of FDP as a single unit to flex the tip of the fingers and if one finger FDP is affected, the other fingers cannot flex fully. FDP tendon to ulnar 3 fingers share the same muscle belly thus any block in FDP action in one of the ulnar 3 fingers the other two cannot flex.

Swan-neck Deformity

There is hyperextension of PIPJ and flexion of DIPJ. It is caused by dorsal subluxation of the

lateral band following FDS dysfunction (rheumatoid hand or lacerations) or volar plate injury.

Boutonnière Deformity or Buttonhole Deformity

There is flexion of PIPJ and hyperextension of DIPJ, resulting from central slip rupture and volar subluxation of lateral bands. It can be due to injury or inflammation.

Camptodactyly

There is flexion deformity of PIPJ, usually of the little finger. It is hereditary and is painless and progressive. In children the deformity disappears when the wrist is flexed but after adolescence the deformity increases rapidly and becomes fixed by contracture of skin and ligaments.

Clinodactyly

There is radial deviation of terminal phalanx of 5th digit. It is familial and characterized by normal bone structure and no periarticular swelling.

Trigger Finger

Triggering of fingers, commonly ring finger is due to difficulty of flexor tendon to negotiate into A1 pulley of fibrous flexor sheath. This may be due to nodular thickening of the FDS tendon or narrowing of the opening of the fibrous flexor sheath. Patient typically develops difficulty in bending and straightening the finger with sudden painful unlocking at the base of the finger on active extension after a catch.

It can be due to repetitive strain or inflammatory disease or diabetes or congenital as in the thumb (30% spontaneous recovery by the age of 1 year).

Dropped Finger

It can be due to rupture of extensor tendon due to laceration, paralysis (radial or posterior interosseous nerve palsy), subluxation of extensor tendon over the ulnar side in rheumatoid arthritis or rupture at wrist (little or ring finger) due to ulnar head subluxation in rheumatoid (Vaughen Jackson syndrome). The intactness of extensor tendon can be confirmed either by tenodesis test, dropped finger extends on palmar flexion of the wrist or by asking the patient to maintain extension after passive correction.

Jersey Finger

Rupture of FDP tendon from insertion at distal phalanx usually involving ring finger in sports injuries or shirt pulling injury.

Snapping Tendon

On flexing the finger patient may notice snapping of extensor tendon over the MCPJ. It is due to disruption of juncture tendon or saggital bands.

Giant Cell Tumor of Tendon Sheath

Isolated solid swelling especially over volar aspect of flexor tendon sheath of the base of the finger.

Acromegaly

Disproportionately large thick set, stoicky hand is suggestive of acromegaly. One must look for prognathism.

Dupuytren's Contracture (Fig. 8.7)

It is proliferative fibrositis involving the palmar aponeurosis of hand, resulting in puckering of skin, nodular thickening and may progress to fibrous longitudinal bands.

History of onset, progression, previous surgery for same problem, anticonvulsant medication, family history and functional disability are important.

a. Look at both hands for presence of flexion deformity usually of MCPJ, PIPJ and rarely DIPJ of little and ring finger, nodular thickening or puckering of skin in the palm.

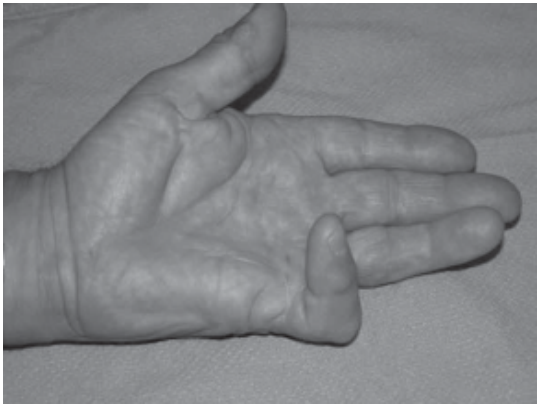


Fig. 8.7: Dupuytren's contracture little finger

b. Look at dorsum of hand for knuckle pads, which is usually on one PIPJ.
c. Dupuytren's diathesis-familial predisposition of some patients with multiple areas of involvement and early onset of the disease. Look for Peyronie's disease (chordee), plantar fibromatosis and knuckle pads.
d. Feel for tenderness (usually painless).
e. Active and passive movements of fingers.
f. Distal neurovascular examination.
g. Heuston's tabletop test is used to decide when to operate. The patient is asked to keep the palm flat on the table and if the patient's palm cannot touch the flat surface of the table evenly because of the flexion deformity of the finger, it indicates the severity of flexion contracture and the need for surgery.

EXAMINATION OF THE HAND WITH LACERATIONS

Mechanism of the Injury

The injury and anatomical structures at the site of lacerations must be thoroughly examined. Palmar lesions involve flexor tendons and neurovascular structures; dorsal lesions usually involve extensor tendons, bones and joints. Axial and rotational defects provide information about fractures and dislocations. It is important to evaluate the degree of contamination.

For example, laceration at the volar aspect, base of middle finger, (Zone II) due to holding a knife with a clenched fist can result in injury to skin, nerves, blood vessels, FDS, FDP, and sometimes bone. The level of flexor tendon laceration will differ on extension of the fingers. The FDP laceration is more distal to FDS laceration; this can be easily missed if we do not know the position of the hand at the time of injury.

Skin

Assess the extent of the damage, presence of skin loss and soft tissue damage and need for any local flaps.

Vascularity

This is assessed as discussed by Allen's test, both at wrist and fingers.

Tendons

Test the anatomical sites for tendons that are liable for injury at that spot. Visible laceration in the tendon, more than 30-40 percent of the substance needs exploration. Division of both flexor tendons to a digit result in pointing sign.

Nerves

i. *Sensory examination:* Check for touch using a cotton swab or the tip of your finger and pinprick sensations for any nerve damage. Running the ballpoint pen barrel across the digit can make out lack of sweating distal to the site of injury. In a normal finger it will adhere and drags, in denervated digit the pen runs smoothly. Fingertip should be examined for two-point discrimination, less than 6 mm is normal but it varies with age.
ii. *Motor examination* for muscles supplied by the nerve.

Skeletal System

Examine for fractures, open joint injuries and dislocations.

In examining a major crush injury of the hand it is better to give regional anesthesia to make the patient comfortable for assessing the extent of the damage and for debridement, but always check for nerve injuries and distal vascularity before hand.

In treating major injuries, the order of importance is:

1. Circulation, reduction of dislocation or realigning the twisted finger.
2. To provide skin cover.
3. Adequately align bones and joints.
4. Tendon and nerve function.

Partial transection of the vessel can result in life-threatening bleeding; no blind clamping should be done as it may injure the nerves.

High-pressure injection injuries (industrial accidents, lead paint) may present with very small punctured wound, but this needs aggressive treatment with extensive debridement and wound should be left open.

Late presentation with healed laceration, one should examine for damage of tendons, nerves, vessels, bones and adjacent joint stiffness.

Reflex Sympathetic Dystrophy or Complex Regional Pain Syndrome (Types I and II)

Reflex sympathetic dystrophy is a neurological dysfunction characterized by burning pain, swelling, stiffness and discoloration due to vasomotor disturbance. The pain can be out of proportion to the nature of injury. It goes through three phases.

Stage-i Acute phase (0 to 3 months) characterized by swelling, pain, warmth and stiffness. X-ray can be normal, triple phase bone scan shows increased uptake in early and late phases.

Stage-ii Dystrophy (3 to 6 months) characterized by glossy skin, change in pain, cool, contractures.

Stage-iii Atrophic (6 to 9 months) characterized by tight skin, contractures and diffuse punctate osteopenia on X-ray.

TRAUMATIC AMPUTATIONS AND MICROSURGERY

Assessment and patient selection is important by clinical judgment for replantation. Patients should be referred to appropriate microsurgery center if they satisfy the criteria.

Nature of Injury

Sharp amputation is better than crush or avulsion injuries. Multiple level cuts are unsuitable for replantation.

Level of Injury

Better results are with distal forearm to hand amputations. Amputation at proximal phalanx level leads to poor flexor tendon function.

Age

Children do better. Very elderly with not much demand, need not have replantation.

Indications

Multiple digit injury, thumb amputation proximal to IP joint, amputations in children, clean amputations at hand, wrist or distal forearm.

Absolute Contraindications

Severe injuries or associated medical problems, multiple level injury of the amputated part, refusal to abstain from smoking for at least 3 months postoperation, and psychotic patients.

The amputated part should be transported in a cool pack with no direct contact with the ice.

HAND INFECTIONS

Paronychia

It is the infection of the nail bed. Usually inside the nail fold, characterized by pain, swelling, and redness.

Felon

Subcutaneous abscess of distal pulp.

Suppurative Flexor Tenosynovitis

Suppurative flexor tenosynovitis is the infection of the flexor tendon sheath. It presents with Kanavel's four cardinal signs—pain on passive extension, finger in flexed position, severe tenderness along the tendon sheath and sausage digit. The infection can spread into the deep spaces—small finger infection can spread to ulna bursa, index and thumb infection can spread to the thenar space, middle, ring and small finger infection can spread to the midpalmar space.

Thenar Space Infection

Patient will have pain, swelling over the thenar eminence with painful flexion of the thumb and the index finger.

Midpalmar Infection

Results in loss of midline contour, pain on movement of middle, ring and little finger.

Web Space Infection

Web space infection can result in web space inflammation, with widening and collar stud abscess.

Human Bite

This results in serious soft tissue and bone infection, more common after punching injuries over knuckles, especially III and IV MCPJ. The wound may be deceptive, and should be explored in the theater and debrided.

9 CHAPTER Examination of Peripheral Nerves and Brachial Plexus

The presentation of peripheral nerve problem can be of deformity, loss of function, neurological symptoms and sometimes neuralgic pain. The onset, nature of any injury, progression and other relevant histories must be documented as discussed before. Patient's expectation must be known. Neurological examination is done by testing individual components of motor, sensory (sympathetic and cutaneous sensation) and reflex changes to identify the level of lesion.

Cut injury wounds or other injuries causing nerve damage should be examined for associated tendon or muscle damage, vascular damage and bony involvement in addition to contamination.

Expose the patient fully rather than exposing only the involved region.

For example, a nerve palsy of hand could be due to cut injury or surgery in the neck. Patient should be undressed to expose the whole of upper arm and the neck to look for any scars or wounds.

LOOK

Attitude of the Limb

Example: Erb's palsy patient may keep the arm in "Waiter's tip position" **(Fig. 9.1)**.

Deformity

Example: Clawing in ulnar nerve and median nerve palsy or isolated ulnar nerve palsy. Wrist drop or fingers drop in radial nerve palsy **(Fig. 9.2)**. Foot drop in lateral popliteal nerve palsy.

Trophic Changes

Pulp atrophy, loss of hair, loss of sweating, trophic ulcers.

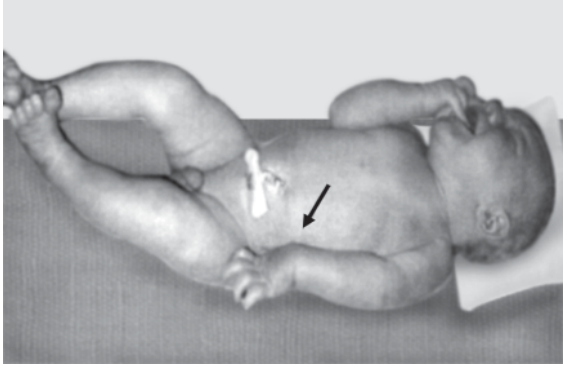


Fig. 9.1: Erb's palsy

Fig. 9.2: Wrist drop

Muscle Wasting

Profound wasting of denervated muscles in chronic condition.

Condition of Skin and Soft Tissues

Healed scars, cuts or punctured wounds along course of nerve, hypopigmentation (Hansen's), café-au-lait spots and multiple swellings—neurofibromatosis.

Associated Bony Injury

Example: Middle third humerus fracture can cause radial nerve palsy, supracondylar humerus fracture can cause radial, median or ulnar nerve palsy, proximal tibial or fibular neck fracture can cause common peroneal nerve palsy, knee or hip dislocation can cause damage to sciatic nerve.

Dysmorphism

Local gigantism (neurofibroma); limb hypoplasia—neurofibromatosis causing pseudoarthrosis tibia (look for neurocutaneous markers); Hansen's disease causing resorption of digits, dysmorphic face, deformities due to involvement of peripheral nerves; Neuropathic joints (foot or ankle) causing disfigurement and distortion of joints and foot ulcers.

FEEL

Ask for tender spot before palpation and always look at patient's face while palpating for tenderness.

Warmth

The paralytic limb is susceptible to the environmental temperature changes. Periphery of the paralytic limb may feel less warm than the other side.

Tenderness

The most tender spot should be palpated last. Assess for soft tissue or bony tenderness and the most tender area. The trigger spot causing burning or sharp pain distally due to nerve irritation can be identified. Tenderness along the course of the nerve should be sought.

Swelling

Nerve swelling or thickening should be felt for in any neurological symptoms. The nerve swelling (neurofibroma) is usually oblong, soft to firm in consistency, can be multiple, moves perpendicular to the long axis of the nerve and has little movement along the long axis of the nerve. Neuritis (Hansen's–lepra reaction) results in diffuse swelling along the course of the nerve with evidence of inflammation. Palpable cutaneous nerves are ulnar nerve at elbow behind the medial epicondyle, and common peroneal nerve at the fibular neck. These nerves can be thickened in Hansen's disease and hypertrophic neuropathy. Proximal neuroma and distal glioma can be felt at the site of untreated long-standing nerve injury.

Provocative Test

a. *Tinel's sign:* Percussing the nerve along its course from distal to proximal can identify any bare nerve ends (without myelination). It is a helpful sign to assess the progression of nerve regeneration. On percussion, a sharp shooting or tingling pain occurs along the distribution of the nerve due to nerve irritation or it can be a localized electric shock like sensation at the site of percussion due to a neuroma.
b. *Direct compression of the nerve:* To reproduce the nerve symptoms by direct pressure on the nerve in different conditions like direct carpal compression test in carpal tunnel syndrome and direct tarsal compression test in tarsal tunnel syndrome.

MOVE

Active movements of the joints should be tested first. Patients with full range of active movements need not be tested for passive movements.

If there is restriction then it could be due to pain, fixed deformity, loss of muscle power or mechanical block. This warrants testing for passive movements to identify the range of movement and the probable cause. Assessment of free movements of the joint is mandatory before considering tendon transfer. Stability of the joints must be assessed.

Trick movements should be identified.

Example: For active abduction of shoulder in supraspinatus paralysis, patient may sway the body to the same side to get initial abduction and then with the help of deltoid can carry out rest of abduction.

Example: In radial nerve palsy the finger extension at IPJ may mimic extension at MCPJ (interossei is responsible for IPJ extension, supplied by ulnar nerve while MCPJ extension is by long finger extensors, supplied by radial nerve).

Subluxation of the nerve especially ulnar nerve at elbow should be felt for on flexion/extension movements.

NEUROLOGICAL EXAMINATION

Motor Examination

a. Bulk
b. Tone
c. Power—Assessed by MRC grading
   - M0– No active contraction.
   - M1– A flicker of contraction seen or found by palpation.
   - M2– A weak contraction which can produce movement with gravity eliminated by positioning of the limb.
   - M3– Movement against gravitational resistance.
   - M4– Movement against gravity and some resistance.
   - M5– Normal power.

Sensory Examination

Fine touch and pinprick assessed in autonomous zones of the nerve distribution. Two-point discrimination at fingertips, vibration and joint position sense are assessed.

Autonomic functions tested are sweating (pen tip or starch–iodine test) and pilomotor erection.

Reflexes

Superficial reflex 'polysynaptic reflex arc'.

Abdominal reflex—upper or lower quadrant, cremastric reflex, bulbocavernosus reflex, anal wink reflex, plantar reflex.

Deep reflex 'monosynaptic reflex arc'.

Biceps, triceps, supinator, knee, ankle jerk.

EXAMINATION OF INDIVIDUAL NERVES

MEDIAN NERVE

Median nerve (C 5,6,7,8,T 1) is formed by the union of medial root of medial cord and lateral root of lateral cord. It supplies the flexor muscles of the forearm (except flexor carpi ulnaris and ulnar half of profundus), thenar muscles – abductor pollicis brevis, opponens pollicis, superficial head of flexor pollicis brevis; first and second lumbricals. The anterior interosseous nerve is a branch of median nerve and it supplies the deep flexor compartment, flexor pollicis longus, flexor digitorum profundus radial half and pronator quadratus. This branch ends by supplying the front of wrist and distal radioulnar joint. The median nerve is responsible for sensation over the volar aspect of lateral three and half digits.

Motor Examination

To test muscle power it is easy to keep the patient in the position of muscle action and ask the patient to hold in that position. The examiner should resist this position by opposite movement. This tests the power of the muscle.

The other way is the patient should perform the action of the muscle against resistance of examiner's hand. This is sometimes too cumbersome if the patient does not understand the direction of action of the testing muscle. *(Please note when a patient does the movement against resistance it*

is in the direction of primary action of the muscle, when an examiner tests against resistance opposite force is applied to the function of testing muscle).

Long flexors: Flexor digitorum superficialis (FDS) and profundus (FDP). FDS is tested by holding the fingers in extension, the finger to be assessed is asked to bend **(Fig. 9.3)**. Flexion occurs at PIP joint due to the action of FDS. This test can be deceptive in little finger, because of normal variant.

FDP is tested individually by asking the patient to bend the tip of the finger at DIP joint while stabilizing the middle phalanx.

Flexor carpi radialis (FCR): It is tested by resisted radial flexion at wrist and feeling for the tendon.

Flexor pollicis longus (FPL): It is tested for active resisted flexion at IP joint of the thumb **(Fig. 9.4)**.

Pronator teres and quadratus Patient is tested for resisted supination by the examiner in a pronated and extended elbow for pronator teres. Pronator quadratus is tested in a flexed elbow.

Abductor pollicis brevis (APB): The APB is tested by "Pen test", ask the patient to keep the palm facing up on a table. Hold the pen such a way that the thumb abducts 90° to touch the tip, that is perpendicular to the plane of the palm. Feel for the contracting muscle.

Opponens pollicis: This is tested by asking the patient to touch the base of little finger with the tip of the thumb, feel for the strength against resistance.

Bunnell's "OK" sign: Normally on opposing the tip of thumb, to the tip of index finger, silhouette "O" shape is formed **(Fig. 9.5)**. In patients with anterior interosseous nerve palsy (Kiloh Nevin syndrome) FPL and FDP to index finger is affected causing inability to do "OK", as the index finger tip and the tip of the thumb are hyperextended at IP joints.

Fig. 9.3: Testing FDS

Sensory Examination

It supplies the volar aspect of the radial three and half digits. The sensory supply of the median nerve may be confusing, but usually the volar surface of thumb, index and middle fingers and the dorsal surfaces of the distal phalanges of the index and middle fingers are supplied by median nerve. The smallest autonomous zone of the median nerve is the tip of index and middle fingers.

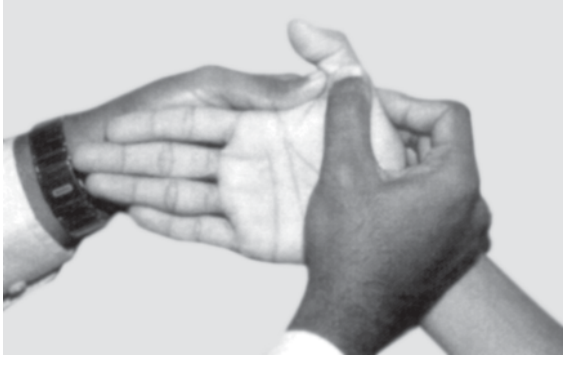


Fig. 9.4: Testing FPL

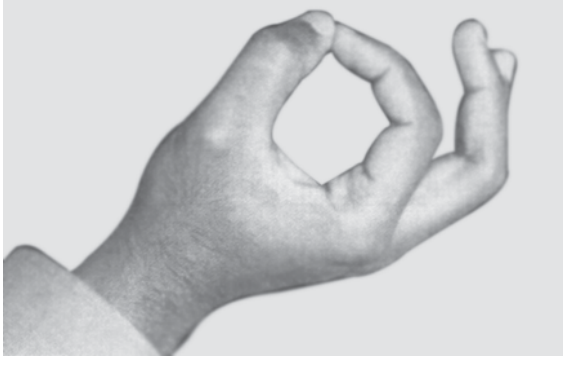


Fig. 9.5: OK sign

Specific Signs in Median Nerve Palsy

1. Wasting of forearm muscles and thenar muscles.
2. Pointing index sign: In high median nerve palsy due to affection of FDS and FDP to the index finger, the index finger cannot be flexed. The other fingers can due to the action of ulnar half of FDP supplied by ulnar nerve **(Fig. 9.6)**.
3. Ape-thumb deformity: Due to inefficiency of the abductor and opponens pollicis, the 1st metacarpal drops to the same plane as the other metacarpals resulting in "Simian hand."
4. Pen test (abductor pollicis brevis): The patient places the affected hand flat upon a table with palm uppermost, a pen is held above the thumb and the patient is told to touch the pen with the edge of his thumb by abducting **(Fig. 9.7)**.

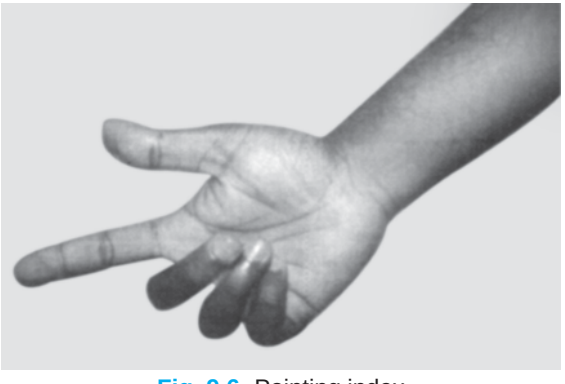


Fig. 9.6: Pointing index

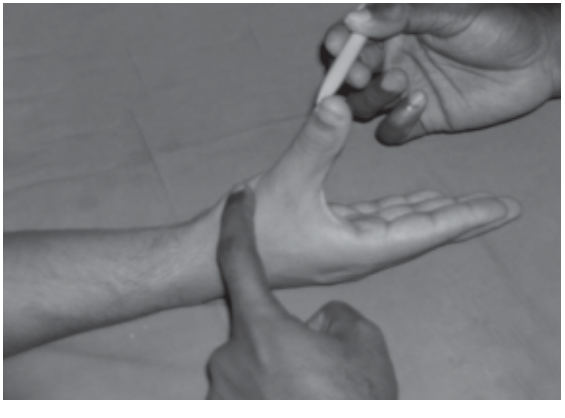


Fig. 9.7: Pen test

ULNAR NERVE

Ulnar nerve (C8,T1) is a branch of the medial cord of brachial plexus. It gives motor branches to flexor carpi ulnaris (FCU), ulnar half of flexor digitorum profundus (FDP), hypothenar muscles—abductor digiti minimi, opponens digiti minimi, flexor digiti minimi, interossei, 3rd and 4th lumbricals, adductor pollicis and sometimes deep head of flexor pollicis brevis.

Sensory distribution is over ulnar 1 ½ fingers and ulnar aspect of hand.

Motor Examination

1. *Flexor carpi ulnaris test*: This is tested by flexion and ulnar deviation of the wrist against resistance and feel the contracting tendon at the wrist and assess its power.
2. *Card test*: Palmar interossei are adductors (Pad), tested by asking the patient to hold a card between the extended fingers and assess resistance against pulling **(Fig. 9.8)**.
3. *First dorsal interosseous*: Place the hand flat on the table with the palm down, ask the patient to actively abduct index finger against resistance, feel for the muscle contraction in 1st web space **(Fig. 9.9)**. Wasting of the muscle may be obvious in 1st web space.
4. *Froment's sign/book test:* To test adductor pollicis ask the patient to grasp a book or card between extended thumb and index finger.

Fig. 9.8: Card test

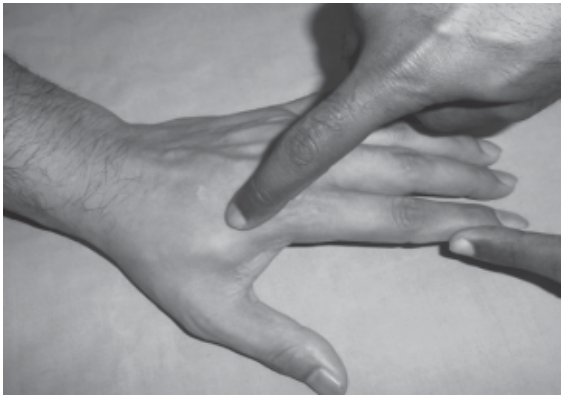


Fig. 9.9: 1st dorsal interosseous test

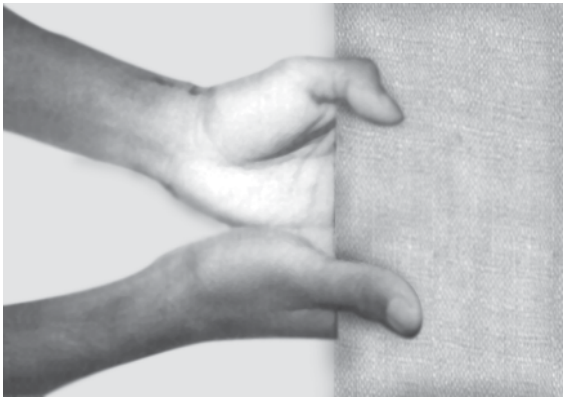


Fig. 9.10: Book test

In ulnar nerve palsy patient tries to grasp the book by flexing the IPJ of thumb (using FPL supplied by median nerve), as the patient is unable to adduct the thumb **(Fig. 9.10)**.

5. *Wartenberg sign:* It is the abduction attitude of little finger due to the unopposed action of extensor digiti minimi (radial nerve) in paralysis of palmar interossei. This attitude becomes more evident on extending the finger and is seen with clawing.
6. *Ulnar claw hand (little and ring finger)*: Clawing is hyperextension deformity of the MCPJ with flexion of the IPJ. This is due to imbalance between flexors and extensors at the MCPJ. The lumbricals and interossei are the prime flexors of MCPJ. Paralysis of these muscles results in hyperextension by long extensors.
7. *Muscle wasting*: Wasting of hypothenar muscles, lumbricals and interossei (guttering).

Sensory Examination

Tested from distal to proximal, especially in the autonomous zone. Altered or decreased sensation in little and ulnar half of ring finger. Loss of sensation over the dorsum and ulnar aspect of hand indicates high lesion as the dorsal sensory branch of ulnar nerve arises 7 cm proximal to the wrist.

Ulnar Paradox

In low ulnar nerve lesion clawing of little and ring finger is very obvious whereas in high ulnar nerve lesion clawing is less and this is due to the paralysis of ulnar half of FDP.

RADIAL NERVE

Radial nerve (C 5,6,7,8 and T1) is a branch of the posterior cord of brachial plexus. It supplies the extensors of the elbow, wrist, fingers and thumb and long abductors of the thumb. Muscles supplied in the arm are triceps, anconeus, brachioradialis, ECRL and ECRB. The radial nerve divides into superficial radial and deep posterior interosseous nerve at the level of lateral epicondyle. Superficial radial nerve passes under brachioradialis and becomes subcutaneous few centimeters above the wrist to supply the dorsum of 1st web space. The posterior interosseous nerve supplies extensor carpi ulnaris, extensor digiti minimi, extensor digitorum longus, extensor indicis, abductor pollicis longus, extensor pollicis longus and brevis, and ends as a pseudoganglion supplying the wrist.

In proximal third extensor aspect cut wound of forearm there can be inability to straighten the fingers or thumb that may be due to posterior

interosseous nerve cut injury or muscle laceration. As abductor pollicis longus and extensor indicis has more distal origin in forearm, if these muscles are functioning it indicates muscle laceration rather than posterior interosseous nerve injury (Rex sign) at that level **(Fig. 9.11)**.

Motor and Sensory Examination

High radial nerve palsy will cause:

a. *Triceps weakness:* This is tested by resisted active extension of the elbow and feeling for the muscle contraction.
b. *Brachioradialis:* Tested by resisted active flexion of the elbow with forearm in midprone position and feeling for the muscle contraction.
c. *Wrist drop:* This is due to paralysis of wrist extensors.
d. *Finger drop:* This is due to paralysis of long finger extensors causing inability to extend fingers at MCPJ (beware of trick movements, as IPJ can be extended by the intrinsic muscles) **(Fig. 9.12)**.
e. *Thumb drop:* This is due to paralysis of extensor pollicis longus and abductor pollicis longus.
f. Sensory loss over the autonomous zone, dorsum of 1st web space.

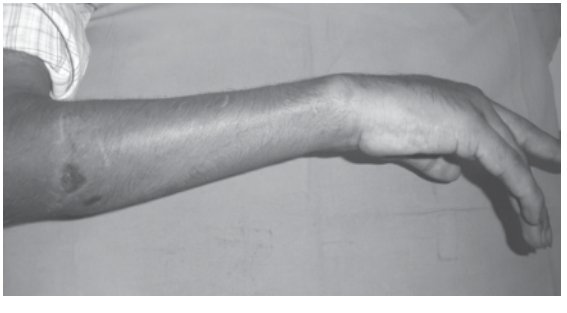


Fig. 9.11: Lacerated wound extensor aspect in proximal third forearm with medial three fingers drop indicates superficial extensor muscle injury without involvement of posterior interosseous nerve

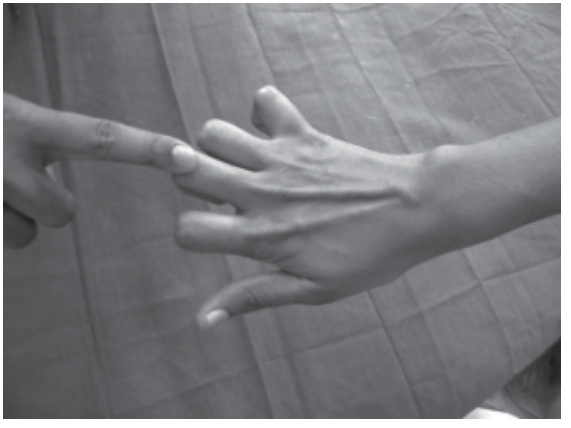


Fig. 9.12: Testing extensor digitorum longus by active extension of MCPJ *(For color version, see Plate 5)*

In posterior interosseous nerve palsy:

a. Dorsiflexion of the wrist results in radial deviation due to paralysis of extensor carpi ulnaris and intact ECRL and ECRB.
b. Finger and thumb drop but no wrist drop.
c. No sensory deficit.

COMPRESSION NEUROPATHY

Ulnar Nerve

Cubital Tunnel

At the elbow the ulnar nerve can be entrapped behind the medial epicondyle due to anatomical or pathological cause.

Anatomical cause—shallow groove, subluxation of the nerve, thick deep (Osborne's) fascia and between the two heads of FCU.

Pathological cause—fracture medial epicondyle, Hansen's neuritis, tardy-ulnar nerve palsy in cubitus valgus.

Findings include Tinel's sign and reproduction of symptoms with full elbow flexion and holding for a minute.

Guyon's Canal

The ulnar nerve can be entrapped as it passes between the pisiform and pisohamate ligament

medially, hook of hamate and insertion of transverse carpal ligament laterally and volar carpal ligament forming the roof.

Causes: Ganglion, ulnar artery aneurysm or thrombosis, fracture hook of hamate, tumors, anomalous muscle, hypothenar hammer syndrome, palmaris brevis hypertrophy.

If the entrapment is more distal in the canal it involves only the motor branches and sensation to ulnar one and half fingers are spared. Sensation over dorsum and ulnar aspect of hand will be normal.

Physical findings include hypoesthesia in ulnar one and half fingers, intrinsic muscle weakness and lack of filling of ulnar artery in ulnar artery pathology (Allen's test).

Allen's Test

Feel both the radial and ulnar artery at the wrist with hand elevated, ask the patient to squeeze the fingers and make a fist. Obstruct the blood flow through the arteries by local pressure. Open the hand and see it looks pale and white, release the pressure on one artery to see the flare. This indicates adequacy of blood supply through that artery. Repeat the same procedure for the other artery **(Figs 9.13A and B)**.

Similar test can be done in the fingers to assess the digital artery flow (Finger Allen's test).

Median Nerve

Pronator Syndrome

Pain in the volar aspect of forearm increases with activity. It can be due to compression of median nerve by ligament of Struther (3rd head of coracobrachialis), lacertus fibrosis, pronator teres muscle or proximal arch of FDS. Patient may have positive Tinel's sign at pronator area, pain on resistance to pronation (in pronator involvement) or pain in the forearm on resistance to isolated flexion of the PIPJ of middle and ring finger (FDS arcade). Nerve conduction test is confirmatory.

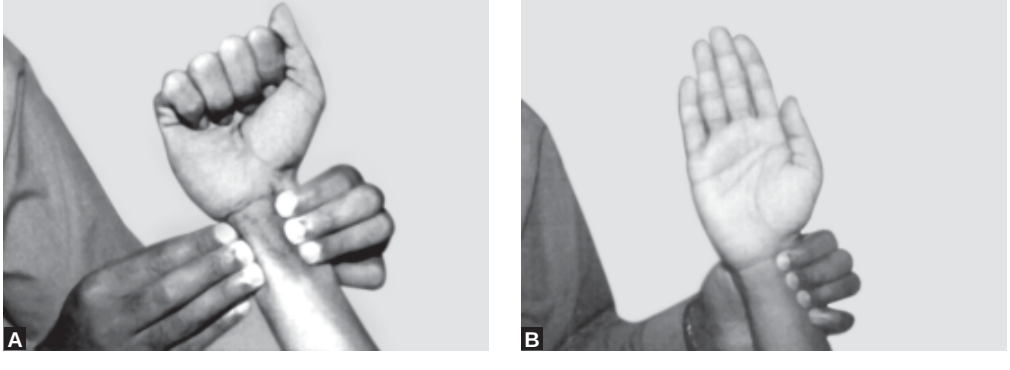
Anterior Interosseous Syndrome

This is usually due to entrapment of anterior interosseous nerve in deep head of pronator teres, which supplies FPL, radial half of FDP and pronator quadratus. This causes forearm pain and inability to make "OK" due to loss of IPJ flexion of thumb and DIPJ flexion of index finger.

Differential diagnosis Mannerfelt syndrome due to rupture of FPL in rheumatoid arthritis.

Carpal Tunnel Syndrome

Discussed in Chapter 7.



Figs 9.13A and B: Allen's test

Radial Nerve

Crutch Palsy

Due to inappropriate length of axillary crutches.

Saturday Night Palsy

Direct pressure of the nerve in the upper arm due to abnormal posture for prolonged time.

Humerus Shaft Fracture (Holstein Lewis Fracture)

Radial nerve is entrapped as it crosses the lateral intermuscular septum in middle third- lower third junction fracture of humerus.

Posterior Interosseous Nerve Syndrome

There is weakness of fingers and wrist due to entrapment of the nerve in proximal supinator (arcade of Frohse), tumors, ganglion, lipoma, radial head fracture or surgery. It manifests with loss of finger extension and dorsiflexion of wrist resulting in radial deviation due to paralysis of ECU. Dorsal wrist pain can occur due to termination of nerve as pseudoganglion at wrist. There is no sensory deficit.

Radial Tunnel Syndrome

This is a syndrome of pain. Radial tunnel is bordered by brachioradialis and brachialis and extends distally to distal border of supinator. Fibrous bands can entrap radial nerve before it reaches supinator, radial recurrent vessels (leash of Henry), arcade of Frohse or by origin of ECRB. This usually presents with pain localized 5 cm distal to lateral epicondyle and aggravated by resisted extension of middle finger (ECRB inserts at the base of 3rd MC) with no motor or sensory deficit.

Differential diagnosis: Tennis elbow.

Cheralgia Paresthetica (Wartenberg Syndrome)

Superficial radial nerve becomes compressed as it comes superficial at brachioradialis and ECRB interval in distal forearm causing sensory disturbance.

Thoracic Outlet Syndrome

Middle aged or young females typically present with neck pain, paresthesia in ulnar nerve distribution with motor weakness due to entrapment of brachial plexus at scalene triangle.

Vascular symptoms like *Raynaud's phenomenon*—intermittent attacks of pallor or cyanosis of fingers and embolic episodes to fingers with gangrene formation can occur. This can be due to cervical rib, anterior scalene muscle contraction or abnormal fibrous band.

Adson's Test

First feel the radial pulse of a seated patient in an externally rotated, slightly abducted arm, and then ask him to turn his head as far as possible towards the affected side and take a deep breath. The test is positive if the pulse becomes feeble or is not felt **(Fig. 9.14)**.

Arm Elevation Test (Roos Test)

Both arms elevated, abducted to 90° and externally rotated with elbows flexed to 90°, now fingers are flexed and extended rapidly for 3 minutes. This causes claudication pain in the forearm and wrist in vascular compromise **(Fig. 9.15)** and inability to complete the test.

Fig. 9.14: Adson's test

Fig. 9.15: Arm elevation test

SUPRASCAPULAR NERVE ENTRAPMENT

Deep diffuse pain in the scapular area and atrophy of supraspinatus and infraspinatus can be the presenting feature. Symptoms can become worse with arm abduction and palpation of suprascapular notch. It can be due to trauma or space-occupying lesion.

BRACHIAL PLEXUS INJURY (FIG. 9.16)

Brachial plexus is formed by the anterior rami of C5,6,7,8 and T1 roots. These roots leave the intervertebral foramen and form the plexus. C5 and C6 roots join to form the upper trunk, C7 continues as the middle trunk and C8 and T1 join to form the lower trunk behind the scalene muscles.

The three trunks then proceed behind the clavicle and each divides into anterior and posterior divisions. The three posterior divisions unite to form the posterior cord, the anterior divisions of the upper and middle trunks unite to form the lateral cord and the anterior division of the lower trunk continues as the medial cord.

Branches from the Roots

1. Long thoracic nerve (C5,6,7) —Nerve to serratus anterior.
2. A twig to phrenic nerve (C3,4,5)—Supplies the diaphragm.
3. Dorsal scapular nerve (C5)—Supplies rhomboids major and minor and levator scapulae.

Branches from the Trunks

1. Suprascapular nerve—Supplies supraspinatus and infraspinatus.
2. Nerve to subclavius muscle.

Branches from the Cord

Medial Cord (Money Makes Many Men Unhappy—MMMMU)

1. Medial cutaneous nerve of arm.
2. Medial cutaneous nerve of forearm.
3. Medial pectoral nerve.
4. Medial root of median nerve.
5. Ulnar nerve.

Lateral Cord (MLL—Major between Two Ladies)

1. Musculocutaneous nerve—Supplies biceps, brachialis and coracobrachialis.
2. Lateral root of median nerve.
3. Lateral pectoral nerve.

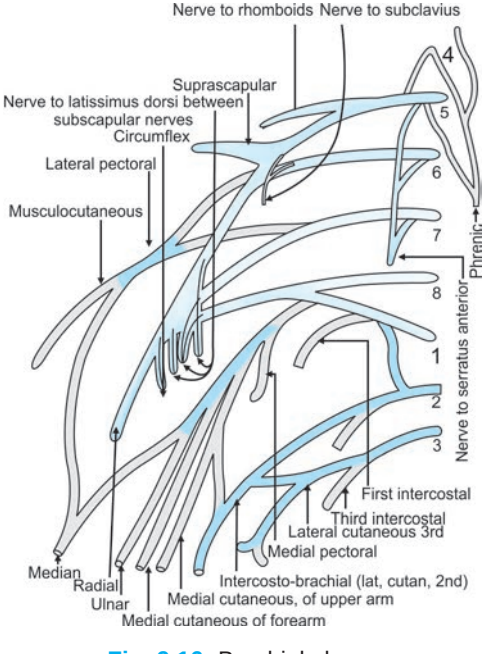


Fig. 9.16: Brachial plexus

Posterior Cord (ULNAR)

1. Upper subscapular nerve.
2. Lower subscapular nerve.
3. Nerve to latissimus dorsi.
4. Axillary nerve.
5. Radial nerve.

EXAMINATION OF THE BRACHIAL PLEXUS

Patient is stripped to the waist to allow visualization of both upper limbs and the dorsal musculature.

Look

Attitude of the Limb

In upper plexus injury (*Erb's palsy*, C5,6 involvement) the limb is typically adducted and internally rotated at shoulder, extended at elbow, pronated at forearm and flexed at the wrist—Waiter's tip posture (see **Fig. 9.1**).

In lower plexus injury (*Klumpke's paralysis*, C8,T1 involvement) the hand functions are primarily affected with paralysis of wrist and finger flexors.

In complete brachial plexus injury the arm is totally flail with no function.

Muscle Wasting

Look for proximal muscle wasting in Erb's palsy, wasting of intrinsic muscles of hand in Klumpke's paralysis and total arm wasting in complete plexus injury. Muscle wasting in the back (latissimus dorsi and trapezius), the scapular region (rhomboids medially, supra- and infraspinatus over shoulder blade) and anteriorly pectoralis major is noted.

Scars

Horner's Syndrome

This occurs in lower plexus injury due to damage of T1 root, which receives sympathetic component from its own ganglion. Horner's syndrome consists of ptosis, miosis, anhydrosis and enophthalmos.

Deformity

There is loss of normal contour with prominent anterior aspect of shoulder, from hooking of acromion and coracoid process. Other deformities at the elbow, forearm, wrist and hand should be noted.

Arm Length Discrepancy

Childhood brachial plexus injury (*Obstetric palsy*) can result in hypoplasia of the limb. In adults it can be due to associated fracture or dislocation.

Feel

Ask for the most tender spot and be gentle in palpation. Start from the cervical spine; feel for any step, tenderness or deformity of spine. Palpate the clavicle, scapula, interscapular area, supraclavicular area and then the shoulder joint for posterior subluxation, elbow and forearm.

Feel the distal pulses.

Move

Examine the active movements of the shoulder, elbow, forearm, wrist and hand. The passive movements are tested to assess fixed deformities or loss of muscle power.

Tinel's Sign

Start from distal to proximal, tapping along the course of the radial, median and ulnar nerves, any tingling sensation or shock like sensation along the nerve is noted and marked. Measurements are taken at regular intervals to assess the motor march. Presence of Tinel's sign indicates infraganglionic lesion.

Systematic neurological examination is done to localize the site of injury —supraclavicular, infraclavicular, supraganglionic or infra-ganglionic injury. In preganglionic lesion —

avulsion of the nerve roots can be associated with spinal cord injury and lower limb involvement, Tinel's sign can be negative and EMG can demonstrate the denervation of paraspinal muscles. The lower root injury can have Horner's syndrome.

Postganglionic lesions can be supraclavicular or infraclavicular. Tinel's sign will be positive. Infraclavicular lesion involves branches from the trunk and below.

Axon reflex test: Presence of triple response indicates intact axon reflex and preganglionic lesion. In postganglionic lesion there is a sequential response of vasodilatation and wheal formation but flare response is absent.

Motor Examination

The examiner stands behind the patient to test trapezius, serratus anterior, rhomboids, supra- and infraspinatus, deltoid and latissimus dorsi. The examiner stands in front of the patient to test pectoralis major and other upper limb muscles.

1. *Diaphragm*: Weakness of diaphragm manifests with breathing difficulty. These patients may have high cervical cord lesion and impaired chest movements and are dependent on abdominal breathing.
2. *Serratus anterior*: Asking the patient to push against the wall tests this muscle. Winging of scapula manifests weakness **(Fig. 9.17)**.

   As patients with brachial plexus injury have difficulty to raise the arm to perform this test one can perform the *scapular protraction* (anterior movement of scapula on thorax) by flexing the arm to 90°and elbow fully flexed, the examiner holds the elbow and the other hand of examiner stabilizes the spine, ask the patient to push the bent elbow forward. If the serratus anterior is weak there is winging of scapula.
3. *Rhomboids*: Ask the patient to place the hand on the hip and to resist the elbow that is being pushed backwards by the patient. Feel for the muscle contraction medial to the scapula.
4. *Internal rotators/External rotators*: Ask the patient to keep the arm by the side of the body with the elbow flexed 90°, apply resistance to the palm while pushing inwards. This tests the subscapularis and pectoralis major (internal rotators).

   Repeat the test asking the patient to resist outward movement. This tests the infraspinatus and teres minor (external rotators).
5. *Subscapularis*: Ask the patient to hold the dorsum of the hand over the buttocks and to lift off the hand (*Gerber's lift off test*) and to push the hand backwards against the examiner's hand. This tests the power of subscapularis.
6. *Supraspinatus* (*Empty can sign*): With the arm in 30° forward flexion and abduction, with full internal rotation and thumb pointing down ask the patient to hold the arm in that position. Push the hand down against resistance and feel for the supraspinatus muscle, sometimes difficult to feel because of the overlying trapezius.
7. *Deltoid*: It is tested by keeping the arm in abduction and pressing down against resistance while feeling the muscle contraction.
8. *Biceps*: The long head of biceps is tested by resisted flexion of the elbow and feeling the muscle contraction in arm.

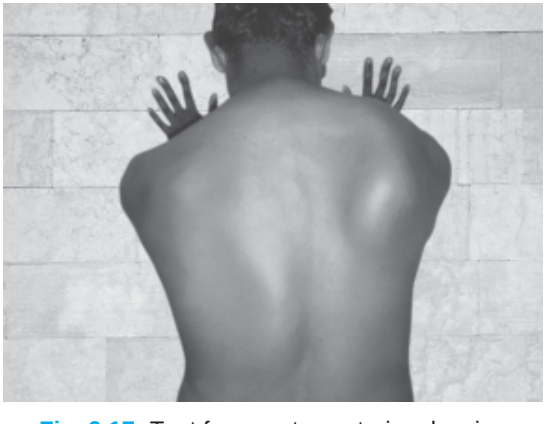


Fig. 9.17: Test for serratus anterior showing winging of right scapula

9. *Brachioradialis*: It is tested by resisted flexion of the elbow in midprone position and feeling the muscle contraction in forearm.
10. *Triceps*: It is tested by resisted extension movement of the elbow and feeling the muscle contraction.
11. *Latissimus dorsi*: Feel the posterior fold of axilla and ask the patient to cough or hold the upper arm in forward flexion of 90° with external rotation and elbow in flexion and resist the extension of arm by holding the elbow (Climber's muscle).

The distal muscles are tested as described in peripheral nerve injuries.

Sensory examination is also helpful to localize the lesion.

C4 root – sensation above the clavicle
C5 root – innervates the lateral arm
C6 root – lateral aspect of forearm and thumb
C7 root – middle finger
C8 root – the little finger and ulnar aspect of hand
T1 root – medial aspect of forearm

LOWER LIMB NERVE INJURY

Sciatic Nerve (L4,5 S1,2,3)

This is formed by the ventral rami of L4,5 S1,2,3. This nerve is injured in stab injury to buttocks, gun shot injury, posterior dislocation of the hip or during hip surgery. Most of the sciatic nerve injuries affect the peroneal component more frequently than complete nerve palsy.

Motor involvement in complete sciatic nerve palsy includes:

Weakness of hamstrings, gastroc soleus.

Tibial nerve component —tibialis posterior, long flexors of toes and small muscles of sole.

Peroneal nerve component—tibialis anterior, long extensors of toes, peroneus tertius, peroneus longus and brevis.

Sensory deficit may be present over the lateral aspect of the leg, dorsal and plantar aspect of the foot.

Common Peroneal Nerve Palsy

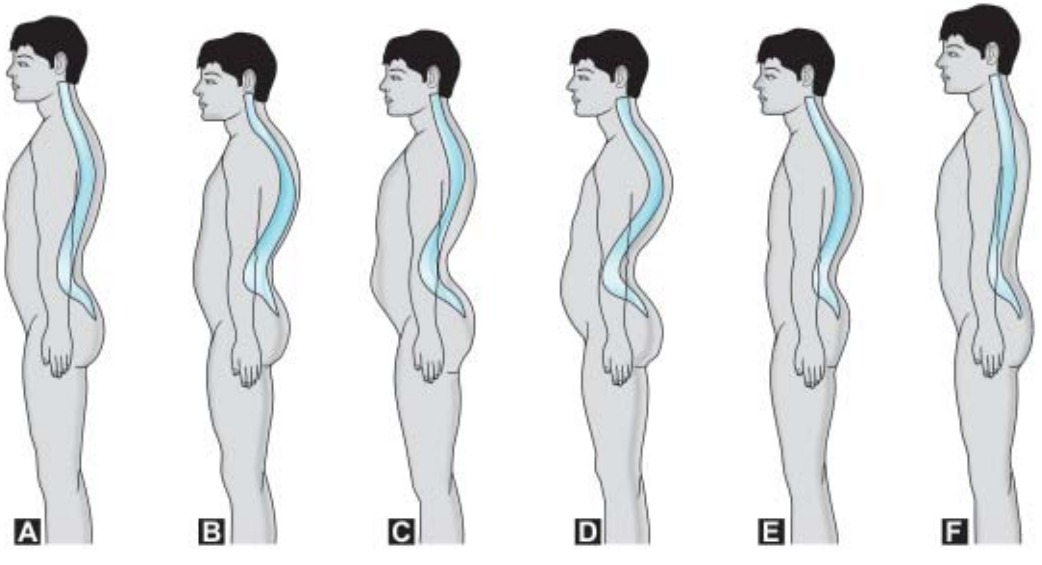
Common peroneal nerve palsy can be a part of sciatic nerve palsy or occurs *per se* in entrapment or fracture of fibular neck from trauma and presents with foot drop (anterior and peroneal compartment component muscles involvement) and absent sensation over the anterolateral aspect of lower leg and dorsum of foot.

10 CHAPTER

Examination of Spine

Normal adult spine has four balanced sagittal plane curves. Thoracic and sacral curves are kyphotic and rigid. These are primary curves present at birth. The cervical and lumbar spines are lordotic and are secondary or compensatory curves. At birth the spine is entirely kyphotic. As infant gains head control and begins to crawl cervical kyphosis changes to anatomic lordosis. As child begins to ambulate, lumbar kyphosis changes to lordosis. Neutral sagittal balance is maintained when a line dropped perpendicular to the horizontal from C 7 vertebra passes through the posterior aspect of the first sacral vertebra.

Back pain and sciatica have affected man throughout recorded history. The oldest surviving surgical text, the Edwin Smith papyrus from 1500 BC, includes a case of back sprain. Today the symptom of back pain is the most common musculoskeletal symptom encountered. The vast majority of sufferers have a minor lumbar sprain or strain, perhaps related to ageing changes in the spine, poor posture **(Figs 10.1A to F)** and lack of physical fitness. The exact pain source is usually difficult to ascertain and exhaustive investigation often is counterproductive, doing no more than reinforcing the sufferers view that there is



Figs 10.1A to F: Various types of posture: (A) Physiologic, (B) Thoracic hyperkyphosis, (C) Lumbar hyperlordosis, (D) Thoracic hyperkyphosis and lumbar hyperlordosis, (E) Total kyphosis, (F) Flat back

something seriously wrong with their back. Early assessment, advice and physical therapy with or without manipulation and the judicious use of analgesics and anti-inflammatory agents will lead to an early resolution of this benign, self-limiting condition in most cases. Chronic cases are more difficult to manage but probably arise more from inappropriate primary management or adverse psychosocial factors than any spinal pathology. The key challenge to the interested practitioner is to identify the more serious cases early and for this, the concept of Red Flags is useful.

CLINICAL RED FLAGS IN BACK PAIN

- Age of onset <20 or >55 years
- Violent trauma, e.g. fall from a height, road traffic accidents
- Constant, progressive, non-mechanical pain, night pain and rest pain
- Thoracic pain—interscapular pain
- Past medical history of malignancy, steroid use or drug abuse
- Systemically unwell or weight loss
- Neurological deficit like cauda equina injury, progressive radiculopathy or myelopathy
- Structural deformity
- Persisting severe restriction of lumbar flexion.
  - Symptoms suggestive of infection
  - Pain persisting more than 8 weeks.

Age

Back pain in children and the elderly arising for the first time should arouse a high index of suspicion and be investigated to rule out infection or tumor.

Occupation

Those in desk bound or driving jobs appear to suffer more from mechanical back pain, as those in very heavy, physically demanding jobs.

Smoking

Smokers have a higher incidence of back pain than non-smokers and poor results with treatment.

Pain

The history of the pain is the most important single part of the assessment. Time must be spent with the patient establishing exactly the date and mode of onset, the exact site and any radiating symptoms as well as the quality (type of pain, e.g. aching, stabbing, electric shock like, burning). Aggravating and relieving factors are of importance.

Mechanical low back pain with radiation is usually posture and activity related, and relieved by rest. This is likely to be due to soft tissue strain or disk degeneration.

Local Pain

Predominant axial pain in the cervical, thoracic or lumbar region involving anatomic structures. In spine the structures innervated to cause pain are muscular, ligamentous structures, fascia, the annulus of disk, bone, the facet joints, dura mater or vascular structures.

Referred or Overflow Pain

All structures of common embryologic segmental origin tend to refer pain in very similar patterns, and the pattern of pain is really determined by the nerve supply to the structure. The end result is that there is substantial overlap between the referral patterns for anatomic structures of the same level such as disk or zygoapophyseal joint pathology as well as the sclerotomal or myotomal referral patterns at many spinal levels. The cervical zygoapophyseal joint pain and its characteristic referral areas were mapped for the site of pain distribution clinically. Pain in the lower limb is less intense than pain in the back. This pain, which is worse in the lumbar area, can be referred to the thighs but not below the knee. This pain is referred along the tissues developed from same sclerotome or myotome with segmental innervations, e.g. discogenic pain from minor disk bulge with no thecal sac or root compression; due to irritation of posterior annulus, posterior longitudinal ligament, facet capsule.

Radiating Pain

Radiating pain indicates involvement of the neurologic structures. Pain along the dermatomal distribution with paresthesia, burning, hyperalgesia or loss of sensation and weakness. This may be due to disk herniation, degenerative process, neural foraminal stenosis, space occupying lesion in spinal canal, intrinsic disease of the spinal cord or nerve root (herpes zoster, neurofibroma). In the lower limbs often associated with paresthesia is most likely due to neural compression. This root pain, which radiates to lower limb below the knee along the dermatomal distribution, is called sciatica. Associated weakness of the involved root can be present.

Claudication Pain

Pain in the buttocks, thigh or calf on walking.

This can be of neurogenic or vascular in origin.

a. Vascular claudication produces pain, which is relieved on standing for few minutes unlike neurogenic claudication where bending forward relieves pain.
b. The pain is relieved quickly in vascular claudication than neurogenic claudication.
c. Cycle test: Patient with vascular claudication gets pain on cycling but not in neurogenic claudication. The spinal canal space increases on bending forwards, for the same reason walking up hill does not cause pain in neurogenic claudication.
d. Palpating the distal pulse may again help to differentiate vascular from neurogenic cause.
e. The claudication distance is fixed in vascular but variable in neurogenic claudication.
f. Cramps and numbness is more common in neurogenic claudication.

Pain at rest, relieved by activity especially bending forwards and pain on extension can be due to facet joint arthrosis.

Localized pain in the back, which is tender on superficial palpation in the paraspinal area, and sometimes with nodularity, can be due to myofacial strain or fibrositis.

Pain from instability can present with extension catch. Flexion of the back is normal but getting up from bent position can produce a catch and excruciating pain.

Onset and Duration

How did pain start in the first place? Sudden shooting pain in the back while lifting heavy weight or straightening the back from bent posture can be from disk prolapse or myofascial strain.

Elderly patient presenting with sudden onset pain after lifting heavy weight can be from compression fracture of osteoporotic spine.

Injuries at work are very likely subjected to personal injury claim and treatment of any kind will hardly improve the symptoms if there is any pending litigation.

Nature of Pain

Electric shock like sensation down the leg starting from the back or well-localized pain radiating from the back is radicular pain due to nerve irritation. Dull pain of poor localisation in the back and thigh area is usually referred pain. Burning pain in the leg again signifies neurogenic origin.

Exacerbating and Relieving Factors

In relation to activities of daily living, rest and night pain, prolonged sitting, bending movements and lifting weight. Coughing and sneezing can produce increased pain in disk prolapse sometimes shooting down the leg below the knee. Simple mechanical back pain is activity related and mostly relieved with rest or lying down posture.

Night Pain

Severe pain can signify infective or tumorous or inflammatory condition of the spine.

Neurological Symptoms

The presence or absence of a neurological pain symptom (e.g. radicular, claudicant) will by now have been established. Similar detail is required with regard to the symptoms of paresthesia (pins and needles), numbness, deadness, weakness, gait disturbance, bladder or bowel symptoms and any other neurological symptoms.

Stiffness

Early morning stiffness of the back and sacroiliac joints can be due to inflammatory spondyloarthropathy. Chest expansion should be checked for in suspected cases of ankylosing spondylitis in young adults. Chest expansion of less than 5 cm signifies restriction due to costovertebral inflammatory arthritis.

Stiffness again can manifest by inability to bend forwards or lean backwards or walking with a "lateral list" (swaying the body to one side on standing and walking). The lateral or sciatic list typically occurs in a posterolateral disk prolapse with root compression where the patient sways his shoulder to the opposite side to relieve pressure on the nerve root.

Miscellaneous

Constitutional symptoms in infection of spine, loss of appetite and weight (malignancy and infection like tuberculosis) and other joint involvement.

Social background, psychological aspect, significant past medical history, family history, menstrual history (in scoliosis to know the age of menarche for assessment of further growth and progression), and treatment history must be recorded.

- What is patient's expectation?
- General examination
- Local examination

THORACOLUMBAR EXAMINATION

Prior to the physical examination you will have noted the patient's gait and posture. Now watch as the patient rises from the chair and observe how much difficulty he or she has undressing. The examination then continues with the patient in underclothes in four positions. These are standing upright, lying supine (the main part of the neurological examination is performed in the supine position), lying prone, and finally with the patient on the side.

Standing Upright (Fig. 10.2)

Observe walking, undressing, abnormalities of posture—alteration of normal curvature. Scoliosis: lateral bending of spine, Kyphosis: increased forward bending of spine, round back, and kyphoscoliosis. Kyphosis may occur in elderly due to osteoporosis, in ankylosing spondylitis in young adults and Scheuermann's adolescent kyphosis. Gibbus: knuckle like localized deformity in spine due to sharp posterior angulation from wedging of one or more vertebrae. This occurs in congenital defect, fracture or spinal tuberculosis. Excessive or diminished lumbar lordosis, furrow sign—deep

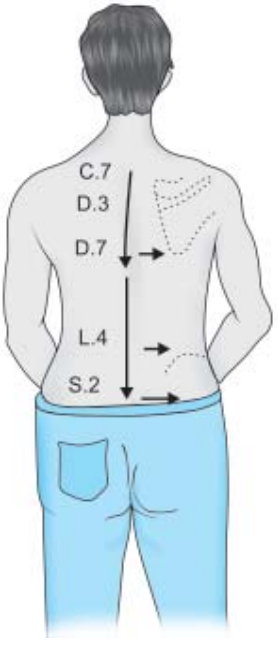


Fig. 10.2: Surface marking of spine

groove between the chest wall and iliac crest because of severe spondylolisthesis causing narrowing of distance between lower ribs and iliac crest, any local abnormalities, paraspinal abscess, neurocutaneous markers like tuft of hair, lipomas, dimple, nevus, café-au-lait spots indicating spina bifida, and scars. Look for the plumb line from base of cervical spine to midline of sacrum. Ask the patient to bend forwards and look for scoliosis and rib hump tangentially (Adam's forward flexion test). Postural scoliosis disappears on flexion. Short leg causing scoliosis disappears when patient sits. Sciatic scoliosis is painful and disappears on treating the underlying cause.

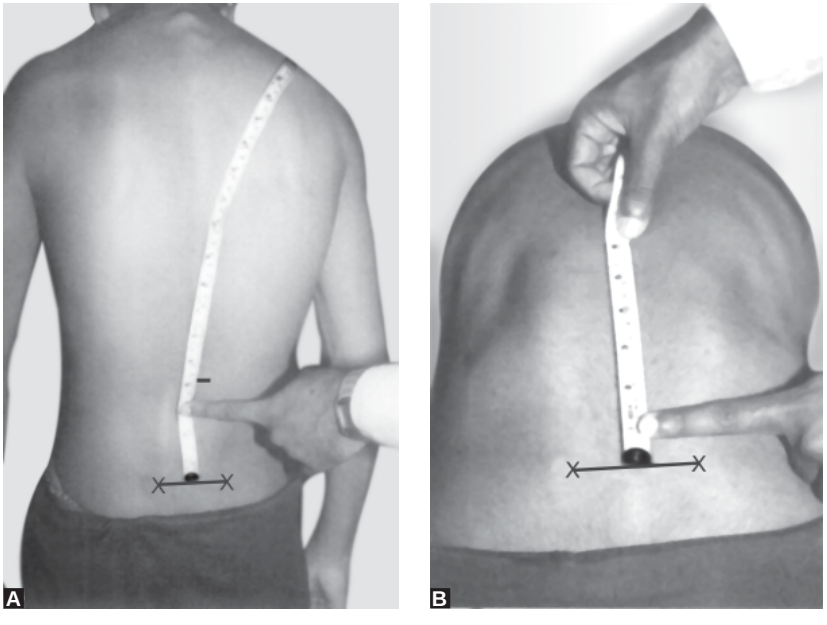
In case of scoliosis look for shoulder asymmetry, tilting of the head, level of scapula, level of pelvis, shortening of leg, neurocutaneous markers, wasted leg, cavus feet and claw toes. Describe the extent of the curve, the apex and the side of convexity; check if the curve is balanced by dropping a plumb line from head to natal cleft. Ask the patient to bend forwards and look for the correctability of the scoliosis and the appearance rib hump. The lateral bending towards the side of convexity can also correct a non-structural scoliosis.

Check Movements

Flexion: Ask the patient to bend forwards, without bending the knee to touch the ground. The distance between the ground and the tip of the hand can be taken as the measurement. Patient with stiff back has limitation. The back muscles may go for spasm. Hip flexion can misguide this movement.

Schober's test is done to identify the flexion at lumbar spine. A point is marked 10 cm above a line connecting the dimple of Venus. The patient bends forwards fully and measurement is taken again. Lumbar spine excursion should be at least 5 cm; less than 3 cm suggests substantial pathology, usually ankylosing spondylitis **(Figs 10.3A and B)**.

Extension catch: Patient getting up from fully bent position experiences catching pain on extension, which may be due to instability or disc problem.



Figs 10.3A and B: Schober's test

Extension: Ask the patient to lean backwards or look at the ceiling by bending backwards. It is difficult to grade.

Lateral flexion: Ask the patient to slide the palm down the thigh and note the distance the hand could reach on the leg on each side **(Fig. 10.4)**.

Extension and lateral bending to side can cause pain in facet joint pathology.

Rotation: Predominantly it happens in the thoracic spine, fix the pelvis and ask the patient to spread the arms out and twist the upper trunk and note the rotations on each side **(Fig. 10.5)**.

Chest expansion: This should be checked in case of suspicion of ankylosing spondylitis; the normal chest expansion is 5 cm or more on deep inspiration.

Feel for localized tenderness in the midline on spinous process, paraspinal area and deep tenderness over loin for kidney pathology. The scoliosis is better appreciated **(Figs 10.6A to C)**. Simple percussion with the examiner's fist can elicit the deep tenderness on the spine.

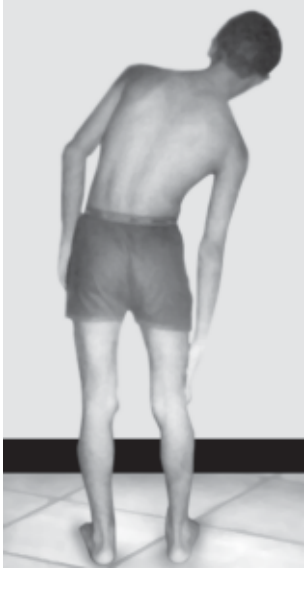


Fig. 10.4: Testing lateral flexion

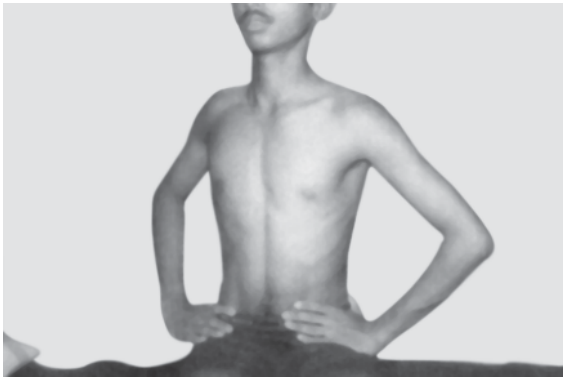


Fig. 10.5: Testing rotation of spine

Standing on tiptoes and heel, it is a quick and easy test to assess power in the lower limb, S1 root for plantar flexion and L 4, 5 roots for dorsiflexion.

Lying Supine

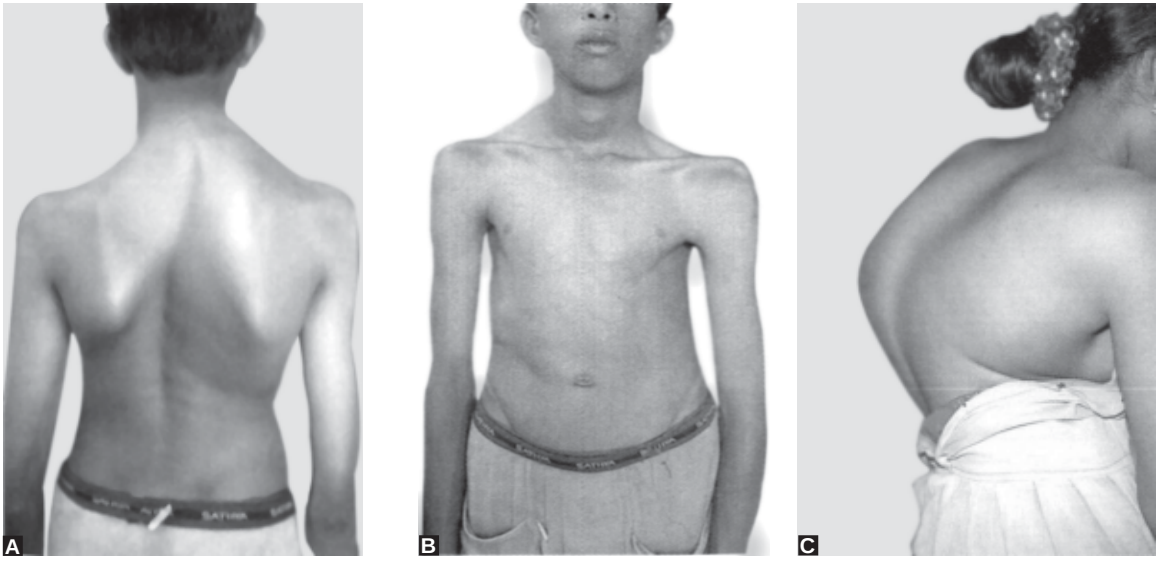
Observe the patient getting onto the couch. Does he overreact with grunting/groaning?

Observe for any gross deformities, swelling, muscle wasting and then feel the abdomen and the pelvis.

Dural Tension Signs or Stretch Tests

Straight Leg Raising (SLR) Test (Fig. 10.7)

The patient is asked to actively raise the leg without bending the knee and assess for pain radiating from the back to the foot and then repeat passively to confirm. The site of radiation is especially asked for. Pain that radiates from back to below the knee on SLR is only considered positive and not just the back pain or thigh pain. The test is considered positive if pain occurs between 30 and 70 degrees of elevation, because there is no true change in tension on nerve roots outside this range. Beware tight hamstrings can cause stretch pain but this does not radiate down the knee. There is normal excursion of sciatic nerve on SLR. If there is a disk prolapse or lateral canal stenosis, this normal excursion is impeded



Figs 10.6A to C: Scoliosis

causing nerve root tension and sciatic pain. Normal L5, S1 root excursion is 4 to 5 cm and less in the upper roots.

If SLR test is positive the leg is lowered down by 10° from the point of sciatic pain and foot dorsiflexed to reproduce the same pain. This stretch test is called Bragaard's test.

Lasegue's Test

Patient in supine position flex the hip and knee to 90°, then keeping the hip flexed, extend the knee. The test is positive if the patient has posterior thigh pain radiating down the leg on extension of the knee.

Bowstring Test

First do the SLRT, then the level at which patient develops pain bend the knee slightly and apply firm pressure with the hand in the popliteal fossa to stretch the nerve. Positive test should produce radiating pain and paresthesia in the leg **(Fig. 10.8)**.

Fig. 10.7: Straight leg raising test

Fig. 10.8: Bowstring test

Well Leg Raise Test or Crossed SLR Test

This is a more definitive test for disk disease causing sciatica. On performing SLR test on the normal leg, the cross over sciatic pain is experienced in the affected leg, usually an axillary disk.

Neurological Examination

Done to assess motor, sensory system and reflexes in upper and lower limbs **(Fig. 10.9)**.

Motor Examination

Bulk, tone and power of the muscles are assessed. A test for each motor root is highlighted by testing the key muscles.

Lower Limbs

L 1,2,3 - Iliopsoas—flexion of the hip
L 4,5 - Gluteus maximus and hamstrings, extension of the hip

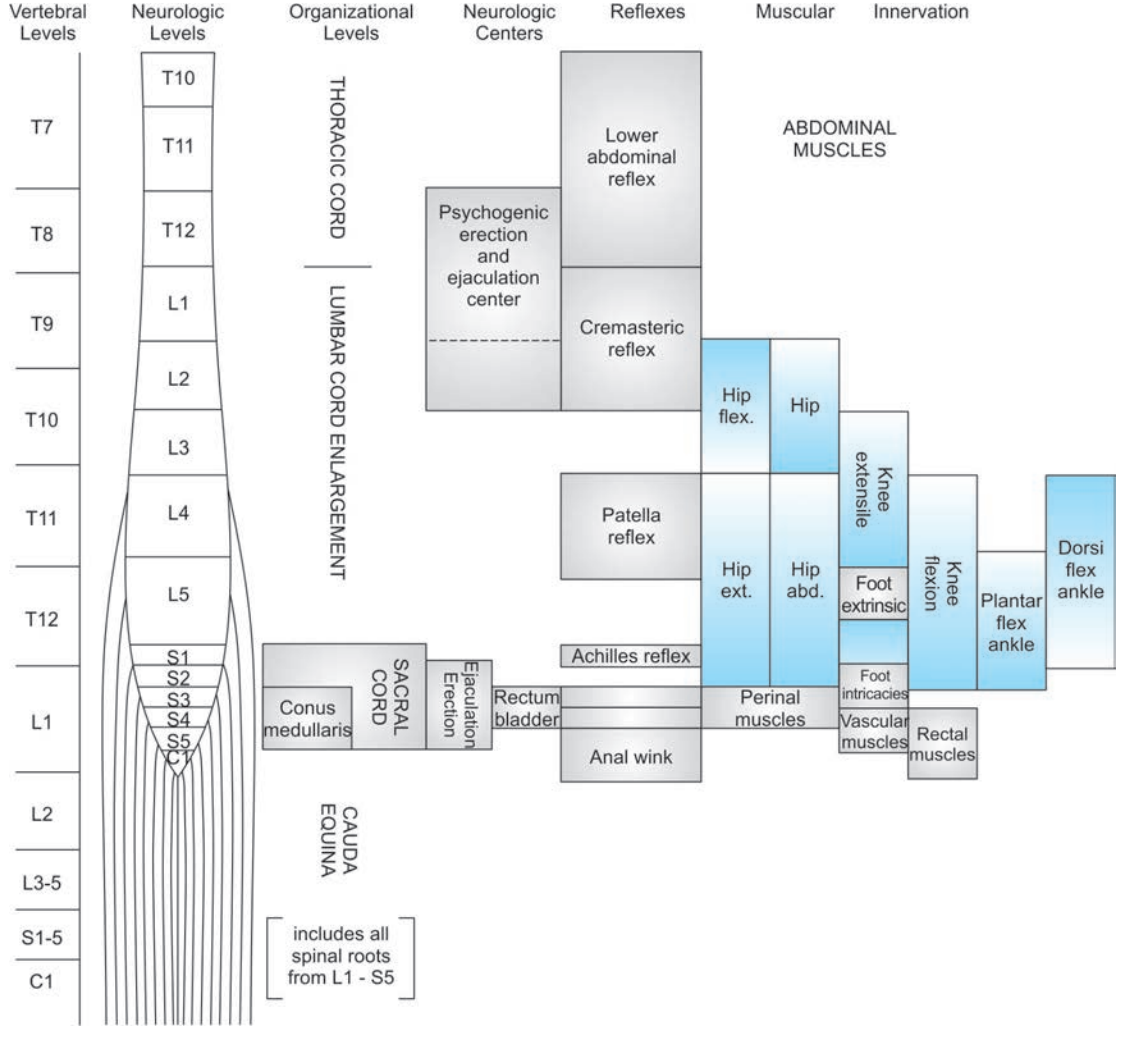


Fig. 10.9: Structural and neurological scheme of vertebral column and spinal cord with neurological implications

L 5 - Gluteus medius—abduction of hip
L 3,4 - Quadriceps—extension of the knee
L5, S1 - Hamstrings—flexion of the knee
L 4,5 - Tibialis anterior and the long extensors of hallux and toes—dorsiflexion of the ankle
S 1,2 - Gastrosoleus—plantar flexion of the ankle
L4 - Tibialis posterior—inversion of the foot
L5, S1 - Peronei, eversion of the foot
L5 - Extensor hallucis longus—dorsiflexion of big toe
S1 - Flexor hallucis longus and gastrosoleus—plantar flexion of toes and ankle

Upper Limbs

C 5 - Deltoid and supraspinatus—shoulder abduction
C 6,7 - Pectoralis major—shoulder adduction
C 5,6 - Biceps—elbow flexion
C 7,8 - Triceps—elbow extension
C 6 - Pronation and supination
C 6,7 - Wrist dorsiflexion and palmar flexion
C 7,8 - Finger flexion and extension
T 1 - Intrinsic muscles of hand—abduction and adduction of fingers

Trunk

Lower thoracic nerve roots motor function is analysed by Beevor's sign. This is a gross test of muscular innervation from the thoracic spine. Patient is asked to do a half situp with knees flexed and the arms behind the head. In normal person because of symmetric contraction of abdominal muscle, umbilicus remains in midline during this maneuver. Root compression or tumor or spinal dysraphism or poliomyelitis results in weakness of the musculature in the dermatome innervated by the root causing umbilicus to deviate towards the strong uninvolved side. This deviation is called Beevor's sign.

Sensory Examination (Figs 10.10A and B)

Lower Limbs

L 1 - over groin
L 2 - over lateral aspect of upper thigh
L 3 - over lateral aspect of lower thigh and front of knee
L 4 - over lateral aspect of leg and front of ankle
L 5 - over 1st web space
S 1 - outer border of foot
S 2 - strip in middle of calf upto sacrum
S 3 - perianal region

Upper Limbs

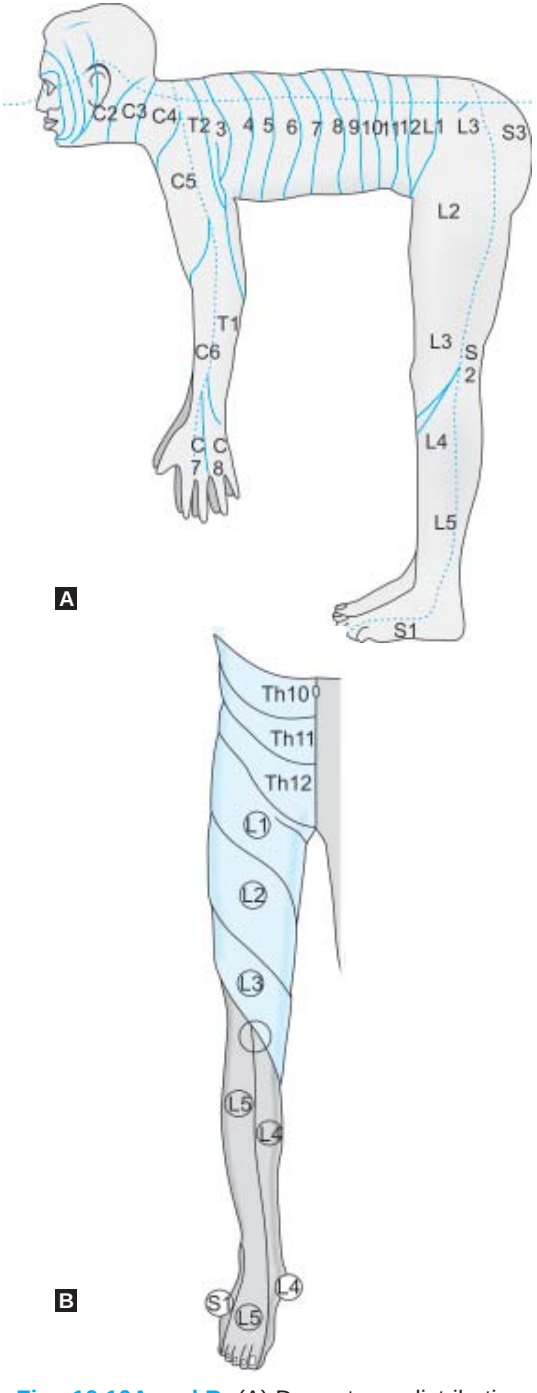
C 4 - supraclavicular area
C 5 - upper and outer aspect of arm
C 6 - radial aspect of forearm, index finger and thumb
C 7 - the middle finger tip
C 8 - ulnar aspect of forearm, little and ring finger
T 1 - inner aspect of arm and elbow
T 2 - inner aspect of upper arm

T 8,9 is upper abdomen, T 10 is umbilicus and T 11,12 is lower abdomen **(Fig. 10.11)**.

Test for light touch and pin prick sensation at the autonomous zone. To find out the level of normal sensation in spinal cord pathology run the finger from below upwards for light touch and pin prick sensation and ask the patient when he or she could feel.

Superficial Reflex

a. *Abdominal reflex:* Stroking the four quadrants of the abdomen in a radial manner beginning



Figs 10.10A and B: (A) Dermatome distribution, (B) Sensory dermatome distribution of anterior leg

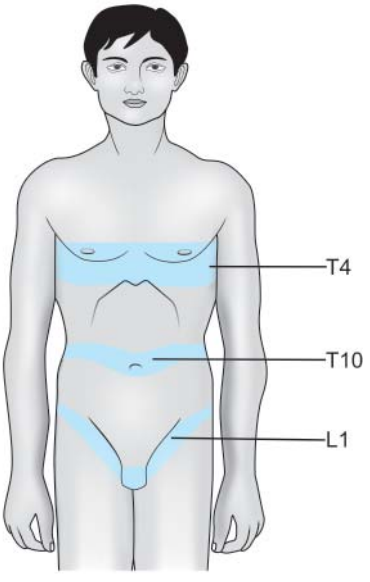


Fig. 10.11: Sensory level surface marking

at the umbilicus to check for contraction of abdominal muscles in each quadrant by movement of umbilicus in the direction of stroke. Absence indicates the level of lesion and spinal cord pathology in scoliosis. D 8,9 is upper abdomen, and D 11,12 is lower abdomen.

b. *Anal reflex*: Stroking or scratching the skin around the anus will cause contraction of the anal sphincter. S 3 and 4 segments are involved.
c. *Bulbocavernosus reflex*: Elicited by squeezing the glans penis or tugging on Foley's catheter and noting the anal sphincter constriction. S3 and 4 segments are involved.
d. *Cremastric reflex*: In males stroking the inner side of thigh causes contraction of detrusor muscle of scrotum. This is absent in L 1 lesion.
e. *Plantar reflex*: Stroking the outer aspect of sole causes plantar flexion of big toe in a normal awake patient. Up-going big toe is called positive Babinski's sign. This is seen in upper motor neuron lesion, unconscious patients, and child below 1 year and cannot be elicited

if sensation over sole is not normal. L5 and S1 segments are involved.

Deep Reflex

Upper Limbs

a. *Biceps reflex (C 5,6)* ***(Fig. 11.12)***: With the elbow flexed and relaxed and a finger over the tendinous insertion gentle tap is given with a patellar hammer. Sudden reflex flexion of elbow due to biceps contraction indicates intact reflex arc.
b. *Triceps reflex (C 7)* ***(Fig. 11.13)***: Tapping over the triceps insertion of a flexed and relaxed elbow produces extension of the elbow due to triceps contraction. This indicates intact C 7 root.
c. *Supinator jerk (C 5,6)* ***(Fig. 11.14)***: With the elbow flexed and forearm in mid-prone position, gentle tap over the radial styloid process causes supination and flexion of forearm due to contraction of brachioradialis.
d. *Hoffmann's sign*: This indicates hyperreflexia in upper limbs. This is demonstrated by flexing the distal phalanx of middle finger and then abruptly releasing it. Positive sign is when the thumb and fingers flex and adduct.

Lower Limbs

a. *Knee jerk (L 2,3,4)* ***(Fig. 11.15)***: Patient relaxed and keeping the knee bent, tapping on the patellar tendon produces a jerk with extension of the knee. It is exaggerated in upper motor neuron lesion and absent in lower motor neuron lesion or with atrophy of quadriceps muscle.
b. *Ankle jerk (L5, S1)* ***(Fig. 11.16)***: In supine position this can be elicited by bending the knee, externally rotating the leg, keeping the ankle in slight dorsiflexion and tapping on the tendo-Achilles tendon. This produces reflex contraction of gastrosoleus with plantarflexion of foot and ankle.
c. *Ankle clonus*: Occurs in upper motor lesion and is pathological if more than 5 beats occur continuously. This is elicited by sudden dorsiflexion of the foot.

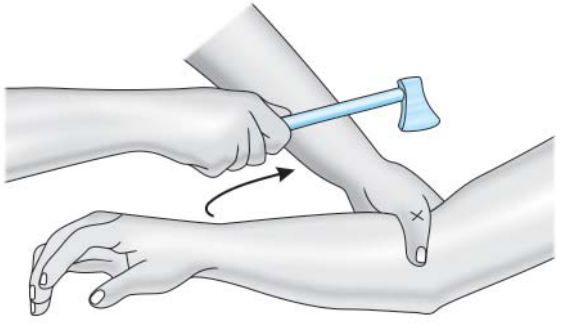


Fig. 10.12: Biceps reflex

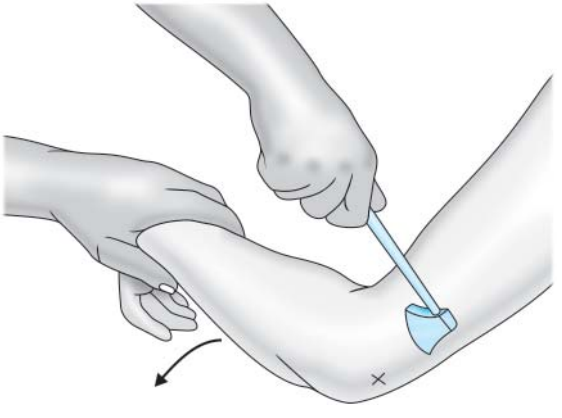


Fig. 10.13: Triceps reflex

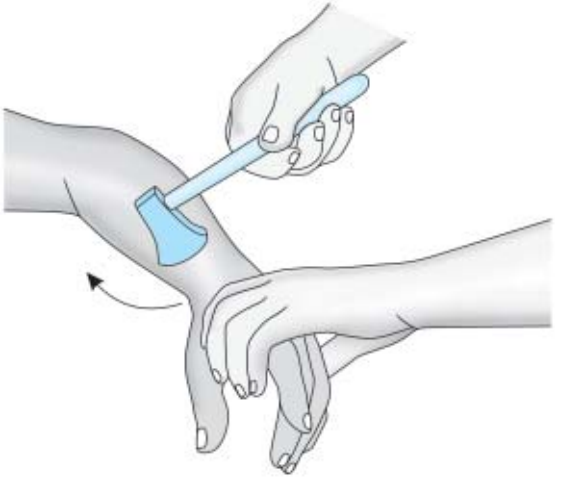


Fig. 10.14: Brachioradialis reflex

d. *Patellar clonus*: Occurs in upper motor lesion and is elicited by sudden downward movement of patella in a relaxed knee to produce beats.

Vascular Examination

Test for dorsalis pedis and posterior tibial pulse, as vascular claudication can mimic neurogenic claudication.

Lying Prone

The presence of gluteal or other muscle wasting may be more obvious now. Feel along the spinous processes for any bump or step. Note tenderness in the midline and three fingerbreadths from the midline in the line of the facet joints. Fibrofatty nodules are common over sacroiliac area, which may be normal if no tenderness is present, and if it is tender to feel, reproducing the same pain, it can be fibrositis.

The femoral stretch test is then conducted to test tension or irritability in the femoral nerve roots (L2,3,4 roots). With the hip extended the femoral nerve is stretched by passively flexing the knee. In a positive test the patient complains of pain in the front of the thigh. Pain can be further increased by hyperextending the hip **(Fig. 10.17)**. The prone position is used to test gluteal (active buttocks squeeze) and hamstring muscle (patient flexes knee against resistance) power. Finally the ankle jerks are best elicited with the patient prone.

Lying on Side

An important part of the neurological examination is to test for skin sensation in the "saddle area" (S3,4) and for the anal reflex (S4,5). Rectal examination can also be performed to test anal sphincter tone. These examinations are mandatory in spinal injury and possible cauda equina syndrome cases. Sacral and coccygeal tenderness (e.g. in coccydynia) is also best assessed in this position.

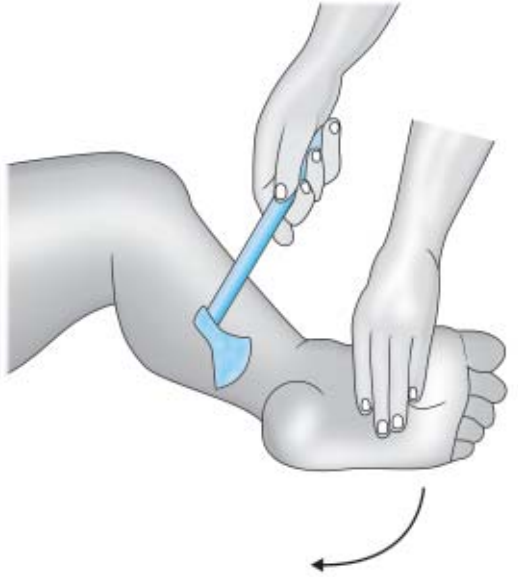


Fig. 10.16: Ankle jerk

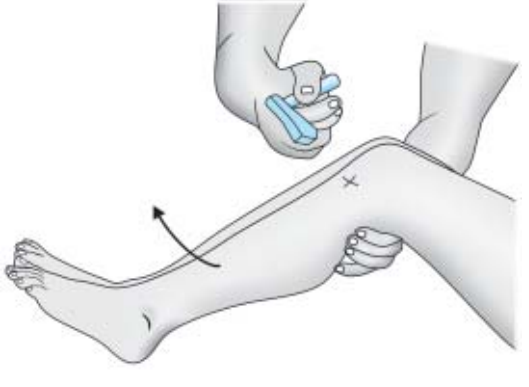


Fig. 10.15: Patellar reflex

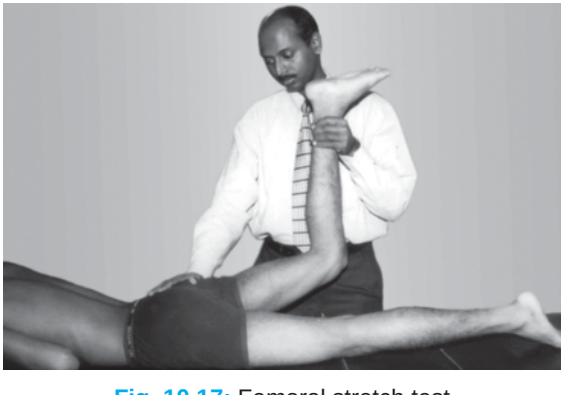


Fig. 10.17: Femoral stretch test

Testing the sacroiliac joints and both hips completes back examination.

Examination after exercise may demonstrate altered neurology in neurogenic claudication due to lumbar spinal canal stenosis.

Waddell's Symptoms and Signs

Symptoms: Pain in the tip of tailbone, complete leg pain, numbness in whole leg and giving way of the leg.

Inappropriate signs: Include the physical signs for non-organic lesion, which helps to identify those patients who have psychological or socio-economic basis for their pain.

1. *Non-organic tenderness:* Superficial tenderness to light touch or widespread tenderness in non-anatomical distribution.
2. *Simulation test:* Low back pain produced by axial compression or rotation of the shoulders and pelvis.
3. *Distraction test:* Patient's attention is distracted to reproduce positive physical findings. Positive SLR test in supine position can be counterchecked by lifting the leg straight with knee extended in sitting position to see whether it reproduces the same sciatic pain.
4. *Regional disturbance:* Like giving way, weakness and sensory loss in a stocking distribution.
5. Over-reaction.

SPECIFIC CONDITIONS

Lumbar Disc Disease

Lumbar disc degeneration is thought to be capable of causing mechanical back pain. This may be secondary to overloading of facet joints, muscular or ligamentous strain at the involved segment due to disc failure or inflammation or annular tear of the disc itself. Pain can radiate to the buttocks and lower limbs but this pain should not be confused with the neurological pain of radiculopathy caused by disc prolapse (protrusion), extrusion (nuclear material breaks through the annulus) or sequestration (nuclear material fragment lies in the spinal canal separate from the rest of the disc). These latter conditions may benefit from surgical discectomy if leg pain persists despite conservative treatment or when neurological deficit progresses **(Table 10.1)**.

Pain is increased on forward bending, lifting weight, on prolonged sitting or on coughing and sneezing. Stretch test and neurological examination are helpful to identify and localize the disease. Most common is the posterolateral disk prolapse at L5,S1 resulting in S1 radiculopathy (the transiting root S1 rather than the exiting root L5) or L4 to L5 disk prolapse resulting in L5 radiculopathy. Acute central disk prolapse with cauda equina syndrome—bilateral buttock and leg pain, bladder and bowel disturbance, saddle anesthesia

Table 10.1: Findings in lumbar disk disease

Level	*Nerve root*	*Sensory loss*	*Motor loss*	*Reflex loss*
L1-L3	L2, L3	Anterior thigh	Hip flexors	None
L3-L4	L4	Medial calf	Quadriceps, tibialis anterior	Knee jerk
L4-L5	L5	Lateral calf, dorsal foot	EDL, EHL	None
L5-S1	S1	Posterior calf, plantar foot	Gastrocnemius/soleus	Ankle jerk
S2-S4	S2, S3, S4	Perianal	Bowel/bladder	Anal wink

(Fig. 10.18) lower motor neuron lesion of lower limb below the level of lesion and absent ankle jerk is a surgical emergency. Far lateral disk prolapse is uncommon and affects the exiting root **(Fig. 10.19)**.

Meralgia Paresthetica

This condition should not be thought of as a lumbar spine condition. Pain or paresthesia is felt in the anterolateral aspect of the thigh in the distribution of the lateral cutaneous nerve of the thigh. The nerve is entrapped or irritated usually at the level of the inguinal ligament.

Marfan's Syndrome

Tall stature with arachnodactyly, arm span more than the height, high arch palate, heart disease, dislocation of lens, scoliosis and pectus carinatum or excavatum.

NEUROFIBROMATOSIS (VON RECKLING-HAUSEN'S DISEASE)

Presents with five or more café-au-lait spots greater than 15 mm in diameter, multiple neurofibromata, local gigantism, axillary freckling, and leg length discrepancy.

Scoliosis

It is lateral bending with rotation of the spine. Scoliosis society has accepted >10 degrees of Cobb's angle as scoliosis. Spinal balance is assessed by plumbline from C7 to know whether it bifurcates the sacrum (natal cleft). List is a pure planar shift to one side in the coronal plane. It may be caused by pain, muscle spasm and more common in lumbar disk prolapse.

Salient Points to be asked are:

- Birth history (cerebral palsy), any congenital problems or other significant past history
- Family history of spinal deformity
- Breathing problem
- Neurological symptoms

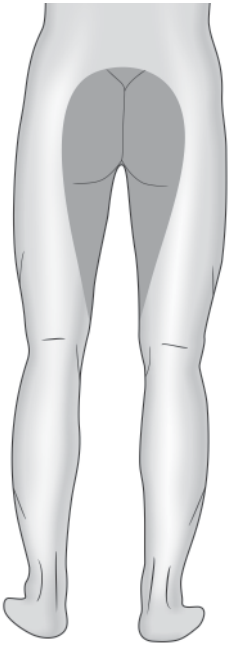


Fig. 10.18: Saddle anesthesia with loss of sensation in dermatomes S3 to S5

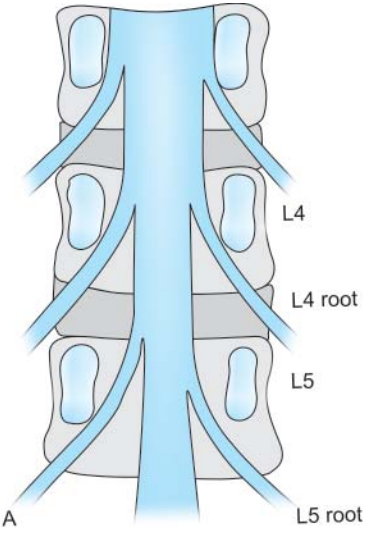


Fig. 10.19: Relationship of exiting and transiting roots

- Deformity—when was it noticed, who noticed it, is it static or progressive balanced or unbalanced, major (primary) and minor (secondary or compensatory curves)

Treatment taken previously.

Aim of examining scoliosis:

1. To know the extent
2. Cause: Idiopathic (Infantile 0 to 3 yr, juvenile 4 to 9 yr, adolescent 10 to 18 yr), congenital, neurological, myopathic, tumors, metabolic, degenerative
3. Structural or non-structural curve
4. Progressive or static; balanced or not
5. Abnormal neurology
6. Pulmonary function

Short, sharp curves with penciling of ribs, scalloping of vertebra and enlarged neural foramen due to dumbbell tumors are characteristic of scoliosis due to neurofibromatosis.

Large "C" shaped curve, more severe, can be of neurogenic or myogenic origin.

Investigation

Long plain X-ray to include both iliac crests for Risser's sign. Scoliosis is most apparent by looking from back, front and sides **(see Fig. 10.6A to C)**. The forward bending Adam's test differentiates the structural from nonstructural scoliosis where the scoliosis persists on bending, with rotation of spine causing rib hump in dorsal spine scoliosis. Lateral bending is to assess the flexibility of the curve (correctability). In a smaller child vertical suspension test can be used to assess the correctability.

Lateral bending film.

Cobb's angle - Angle subtented by drawing perpendiculars from a line along the superior surface and inferior surface of the end vertebrae.

Apical vertebra - Most rotated vertebra at the apex of the curve

End vertebra - Most tilted vertebra towards the concavity of the curve identified by parallel disk space.

Neutral vertebra - Vertebra without any rotation

Stable vertebra - Bifurcation line from mid sacrum cutting the vertebra on the curve equally into two halves. Fusion should be up to this level.

Scanogram of chest wall.

MRI-scan for spinal dysraphism, syrinx, Arnold-Chiari malformation.

Pulmonary function test and spirometry are done and not to operate if vital capacity is below 40 percent in Duchenne's muscular dystrophy.

Kyphosis

Forward bending of spine is called kyphosis. Normally dorsal spine has 30 to 40 degrees kyphosis. Hyperkyphosis is forward bending more than the normality. In thoracic spine >55 degrees is pathological **(Fig. 10.20)**, flexibility can be tested by kneeling test **(Fig. 10.21)**. If the kyphosis is one or two levels then it is called angular kyphosis (Gibbus). If it extends on all spine then it is called rounded kyphosis. It can be congenital, developmental (adolescent Scheurmann's kyphosis—osteochondritis of spine), infection (tuberculosis or nonspecific infection), traumatic, degenerative (osteoporotic compression fracture) and tumors.

Spinal Infection

Spinal infection can be specific infection like tuberculosis or nonspecific pyogenic infection. Pott's spine or caries spine due to *Mycobacterium tuberculi* can occur at any age and usually affects the dorsal spine and can be of multisegment involvement. Commonly, infection will involve the end-plates of two adjacent vertebrae across a disk space. History of exposure to tuberculosis, back pain, night pain, loss of weight and appetite, evening fever, localized tenderness over spine, stiffness and rarely cold abscess formation at

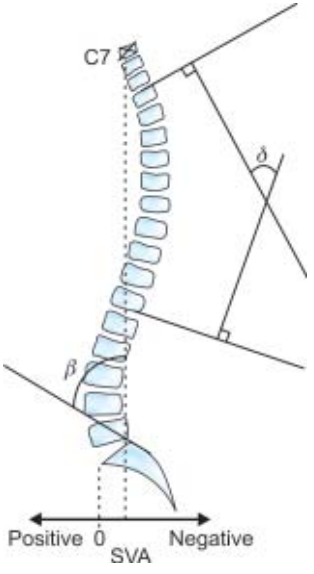


Fig. 10.20: Method of measuring various parameters of sagittal spinal alignment. The sagittal vertical axis (SVA) is positive in kyphotic deformities, because the C7 plumb line anterior to the sacrum. δ = the Cobb angle between two vertebrae. β = the angle of sacral inclination

Fig. 10.21: Kneeling test for evaluating the flexibility of kyphotic deformity

different sites may be present. Paraparesis may be a late presentation.

Pyogenic vertebral osteomyelitis occurs most commonly in lumbar spine and occurs due to hematogenous seedling from genitourinary tract, skin or respiratory tract. The initial focus of infection is the vertebral end-plate. Common presentation is back pain, unrelieved by rest and local spinal tenderness. Constitutional symptoms may be present. Neurologic deficit can occur.

Malignant Spinal Disease

Metastatic disease accounts for the majority of malignant lesions of spine. Elderly patient with sudden onset, back pain without any cause should raise the clinical suspicion of metastasis. There is progressive pain that is unresponsive to rest and normal conservative measures. Previous history of malignancy treatment, loss of weight, appetite, and symptoms and signs of metastasis to lungs and liver must be enquired. The common primary malignancies that metastasize to bones are breast cancer, prostrate cancer, lung cancer, lymphoma, renal and thyroid cancer. Isotope bone scan is helpful to know the extent of metastasis.

Multiple myeloma is the most common primary bone malignancy.

Cauda Equina Syndrome

As the spinal cord ends at lower border of L1 vertebra, any lesion below this level inside the spinal canal can affect only filum terminale (bunch of roots). This is characterized by saddle anesthesia over the genitalia, perineum and buttocks, with retention of urine, disturbance of defecation and lower motor neuron paralysis of lower limbs with absent ankle jerk. A massive central disk prolapse of lower lumbar spine can cause this syndrome and should be treated as a surgical emergency to prevent permanent deficit.

Spondylolisthesis

Forward slipping of one vertebra over another. It is common in lower lumbar spine and can present with back pain or neurogenic claudication pain due to canal stenosis or root pain from lateral canal stenosis and clinically hamstring tightness (limitation of forward flexion), step on palpation of spinous process, flat back and narrowing of loin space (foreshortened trunk) may be noted. Antalgic gait with flexion of knee and prominent buttock can be present in

extreme slips with a "spondylo crisis". Most common is the isthmic type where there is a defect in pars interarticularis, which is developmental and present at young age. The other types are congenital, traumatic, degenerative and pathological in origin.

Inflammatory Spondylitis (Ankylosing Spondylitis)

Pain starts before the age of 40 especially in male of upper back pain with more than 3 months history, insidious onset, and worse in early morning with associated sacroiliitis features alleviated by exercise. Increasing dorsal kyphosis with stiffness with restrictive lung disease limiting chest expansion and severe disease can cause hip arthritis and hip fusion. This results in hunch back deformity **(Fig. 10.22)**, chin to chest deformity **(Fig. 10.23)**, flexion deformity of the hip with fusion of spine obvious in X-ray as bamboo spine.

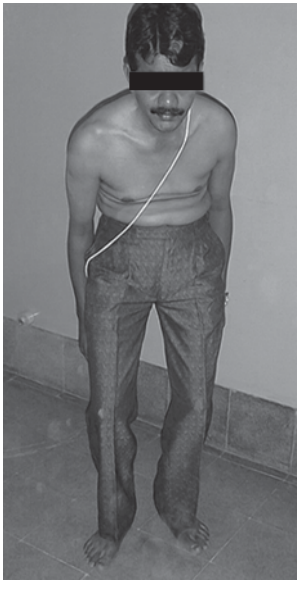


Fig. 10.22: Stooped posture in a young man due to ankylosing spondylitis *(For color version, see Plate 5)*

Scheuermann's Kyphosis

This is a developmental condition of ring epiphyseal osteochondritis of vertebrae occurring in adolescence with increased thoracic kyphosis more common in males presenting with back pain. Radiographic criteria for diagnosis are 3 contiguous vertebral bodies with greater than 5 degrees of anterior wedging, abnormal disk space narrowing, endplate irregularities and Schmorl's nodes, defined as disk herniation into vertebral bodies.

Failed Back Syndrome

Often the result of poor patient selection but other causes include recurrent herniation, herniation at another level, discitis (3 to 6 wk postop with rapid onset of severe back pain), Unrecognized lateral stenosis and vertebral instability. Epidural fibrosis and focal arachnoiditis and nerve damage may due to iatrogenic surgical injury are other causes including wrong level surgery.

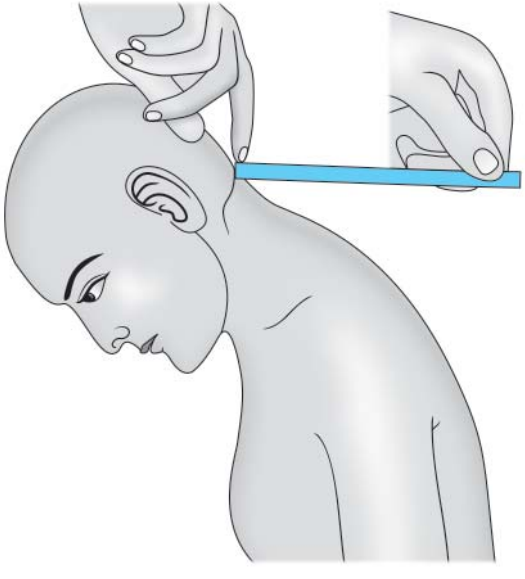


Fig. 10.23: Measurement of the distance between the external occipital protuberance and the wall with the patient standing erect heels touching the wall in cervical kyphosis especially in ankylosing spondylitis

EXAMINATION OF THE CERVICAL SPINE

Neck pain is second only to back pain as a common complaint.

History taking in patients with neck problems must establish details of the following symptoms.

Pain

The mode of onset (spontaneous, sudden, gradually shortly after an injury, etc.) must be established in detail. The site of pain with any radiation, aggravating or relieving factors are important. Neck pain can be in the midline of the neck or in the paraspinal area or can be referred to suprascapular area across the shoulder, and it may radiate to upper limb or the occiput. Patients with severe radicular pain in the upper limb due to cervical disk prolapse may feel better with the arm supported or keeping it raised than letting the arm down to hang by the side of the body. Very localized pain in the supra- or interscapular area can be due to nodular fibrositis in muscle.

Stiffness

This often accompanies pain and may be variable and subjective (often worse in the morning) or constant with objective loss of movements.

Neurological Symptoms

Brachialgia, weakness, numbness, paresthesia (pins and needles sensation) in the upper limbs. Weakness in the lower limbs suggests spinal cord damage.

Deformity

Can be marked cervical flexion deformity from degenerative disease, inflammatory disease or instability; chin to chest deformity in ankylosing spondylitis; wryneck or torticollis (lateral bending) due to various causes.

Dizziness

Sudden neck movements can cause vertebrobasilar insufficiency in cervical spondylosis leading to dizziness.

Miscellaneous

Other symptoms are often volunteered particularly after injuries such as whiplash. These include headache, tinnitus, dysphagia and blurred vision.

Other History

Involvement of other joints, constitutional symptoms, dysphagia, neck swellings, relevant past history, family history, social history and treatment history must be recorded.

What is Patient's Expectation?

Following history-taking, a differential diagnosis should be formed in the examiner's mind. Often the findings on physical examination will make the diagnosis clear without recourse to any special tests. Diagnostic categories include:

Traumatic—whiplash injury, fracture or dislocation with or without neurological involvement.

Degenerative—cervical spondylosis, disk prolapse, canal stenosis, central cord syndrome.

Infective—spinal tuberculosis or other bacterial infection.

Neoplastic—in particular metastatic spinal disease.

Congenital—Klippel Feil syndrome, sprengel shoulder, syringomyelia, cervical rib.

Inflammatory—rheumatoid, ankylosing spondylitis.

Local Examination

As in most orthopedic examinations the look, feel, move system is useful.

Neck examination begins with the patient standing (if he can) and continues with the patient lying supine when the neurological examination is carried out.

Look

The patient should be undressed to allow exposure at least from the waist up.

You must be able to look from the front, side and back of the patient. Some information can be obtained by noting the patient's posture during history taking, observing how much head movement occurs during conversation and whether he has difficulty undressing.

When he is undressed look for asymmetry, muscle wasting—trapezius, deltoid, spinati, pectoral muscles, small muscles of hand; abnormal posture, scars (which may be very inevident) and swellings.

Feel

The cervical spine is palpated from the back with patient sitting and examiner looking at patient's face for any tenderness. Feel the midline of spine for tenderness and deformity, then paraspinal tenderness and then the front of the neck. The anterior aspect of spine can be felt by palpating the spine along the medial aspect of sternomastoid in the upper half from the back of the patient, and carotid pulse can also be felt. Feel only one side at a time to prevent vasovagal shock. Then feel for muscle tenderness or nodular swelling over suprascapular area. Neck swellings are examined as discussed in Chapter 1.

In some patients with muscle spasm, examination of the posterior aspect of the neck may be more easily and reliably done with the patient prone and head resting over a pillow.

Move

Active range of movements are assessed by asking the patient to move and then helping the patient to move to his maximum range without hurting him (assisted active movement). Flexion is by asking the patient to do chin on chest; extension by looking at roof; lateral bending by ear to touch shoulder without shrugging the shoulder and lateral rotation by turning to left and right to look at each shoulder.

Spurling test: This is tested by slight extension, lateral flexion and rotation of the neck to reproduce the same pain in the neck and arm. This manoeuvre causes narrowing of intervertebral foramen causing root irritation in lateral canal stenosis **(Fig. 10.24)**.

Lhermitte's Sign

Presence of electric shock like sensation radiating down into the limbs on flexion of spine either neck or trunk indicates spinal cord lesion.

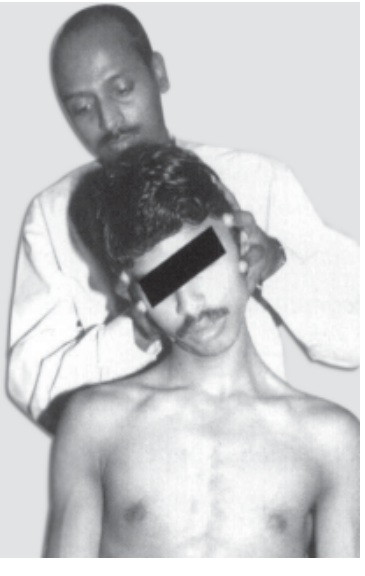


Fig. 10.24: Spurling test

Neurological Examination

Assessment of tone and power in all major muscle groups in all four limbs is required. Reflexes are elicited and sensation is tested (this can be limited to light touch and pinprick in certain circumstances). Of course specific pathologies will lead to specific neurological deficits and the examiner will when experienced enough, be able to quickly identify and demonstrate to any observer the key findings which suggest a specific diagnosis.

Examples are: Cervical disk prolapse leading to radiculopathy. Commonly the C6 or the C7 or the C8 root are involved with a lower motor neurone (LMN) pattern of deficit (weakness, wasting, diminished or absent tendon jerk specific to that root).

Rheumatoid neck, canal stenosis or other pathology such as ununited odontoid fracture can lead to cord myelopathy with more widespread weakness, LMN signs predominating in upper limbs with hypertonicity, brisk reflexes, and upgoing plantar responses (upper motor neuron signs) in lower limbs.

SPECIFIC CONDITIONS

Cervical Spondylosis/Cervical Stenosis

Degenerative disk disease with facet arthropathy of cervical spine can present with discogenic neck pain (mechanical pain), radiculopathy (root compression), and myelopathy (cord compression). In root compression the exiting root is involved, for example, C5, C6 disk disease affects C6 root (remember C1 root exits above C1 vertebra and the other roots are numbered accordingly, C8 root exits between C7 and T1 vertebra) **(Table 10.2)**. Myelopathy is characterised by weakness of upper and lower limbs, sensory disturbance, spasticity, upper motor signs in lower limb, radicular signs in upper limb with weak intrinsics and ataxic broad based shuffling gait. Funicular pain is characterized by central burning and stinging with radiating lightning like sensation down the back with neck flexion (Lhermitte's sign) may be present. Inability to open and close the hand rapidly, little finger abducts from the fingers on asking the patient to keep the finger extended are tests for cervical spinal stenosis. Hoffman's test may be positive where one flicks the terminal phalanx of middle finger into extension suddenly, the index finger and thumb flex. Positive inverted brachioradialis reflex where finger flexion is seen with brachioradialis reflex.

Fibromyalgia

A clinical syndrome of diffuse pain, present at rest and exacerbated by activity, fatigue and sleep disturbance. History of diffuse pain, patient complaining pain all over with tender points on palpation. Pain and tender points can be at insertion of cervical paraspinal muscles on

Table 10.2: Findings in nerve root compression

Level	*Root*	*Muscles affected*	*Sensory loss*	*Reflex*
C3-C4	C4	Scapular	Lateral neck, shoulder	None
C4-C5	C5	Deltoid, biceps	Lateral arm	Biceps
C5-C6a	C6	Wrist extensors, biceps, triceps (supination)	Radial forearm	Brachioradialis
C6-C7	C7	Triceps, wrist flexors (pronation)	Middle finger	Triceps
C7-C8	C8	Finger flexors, interossei	Ulnar hand	None
C8-T1	T1	Interossei	Ulnar forearm	None

occipital condyles, sternocleidomastoid muscle over lateral transverse processes of C5 to 7, belly of trapezius, supraspinatus, over rhomboids, lateral epicondyle, 2nd rib pectoralis insertion, upper outer buttocks and greater trochanter. Some patients have palpable tender knots representing local muscle spasm.

Spinal Cord Injuries

Spinal cord injury (SCI) assessment should not be left to the inexperienced doctor. The first neurological examination in such patients is of vital importance and must be very thorough. Cord injuries may be complete or incomplete and although the former are tragically not rare it is very difficult to be sure that an injury is truly complete on initial assessment. Truly complete cervical cord lesions rarely recover. Incomplete lesions may recover fully. Any sign of cord function below the level of injury means the lesion is incomplete. The poorly understood phenomenon of spinal shock that can last a few days makes a full assessment difficult even for the experienced.

Apart from classifying lesions as complete or incomplete the cervical cord lesions of an incomplete nature can usefully be grouped into anterior, lateral (Brown-sequard), posterior (rare) and central cord syndromes and in any case mixed patterns of injury do regularly occur.

They may be initially in a state of spinal shock that usually lasts 24 to 48 hr and recovery from spinal shock is noted by reappearance of bulbocavernous reflex. On return of bulbocavernous reflex if evidence of complete spinal cord injury is still present, then the patient has a grave prognosis for return of power.

Central cord syndrome: This syndrome is most common in elderly due to hyperextension injury causing damage to central gray matter and surrounding white matter. This is characterized by lower motor neuron lesion in the upper limb due to damage of anterior horn cells causing profound weakness at the level of injury and upper motor neuron lesion in the lower limb with variable sensory disturbance. This syndrome has good prognosis for recovery.

Anterior cord syndrome: This is due to damage of anterior two-third of the cord, sparing the posterior columns. These patients have profound weakness of lower limb than upper limb. The prognosis is worst.

Brown-Sequard syndrome or lateral cord syndrome: This is due to damage of one-half of the cord. This is characterized by lower motor neuron lesion at the level of cord injury, ipsilateral loss of posterior column sensation-position and proprioception, ipsilateral upper motor neuron lesion, and contralateral loss of pain and temperature sensation (due to crossing of lateral spinothalamic tract at each level of entry into spine). This has the best prognosis.

Sprengel Deformity (Fig. 10.25)

Congenital elevation of scapula usually unilateral, producing shoulder asymmetry and small scapula. It can be associated with scoliosis, Klippel Feil syndrome (congenital cervical spine fusion, low hair line and restricted neck movements), fused ribs and other anomalies. Patient usually presents for cosmetic deformity and rarely for restriction of shoulder movements, particularly abduction. Bony omovertebral bar may be present between the superomedial aspect of scapula to lower cervical spine **(Fig. 10.26)**.

Back Pain in Children and Adolescents

In children younger than 5 years the likely diagnosis is tumor or discitis; 5 to 10 years it is Langerhans cell histiocytosis, discitis (tuberculosis or pyogenic) or infective spondylitis, or tumor/leukemia; 10 to 18 years it is Scheuermann's kyphosis, herniated disk, apophyseal ring fracture, spondylolysis or listhesis, ankylosing spondylitis, osteoid osteoma/osteoblastoma in

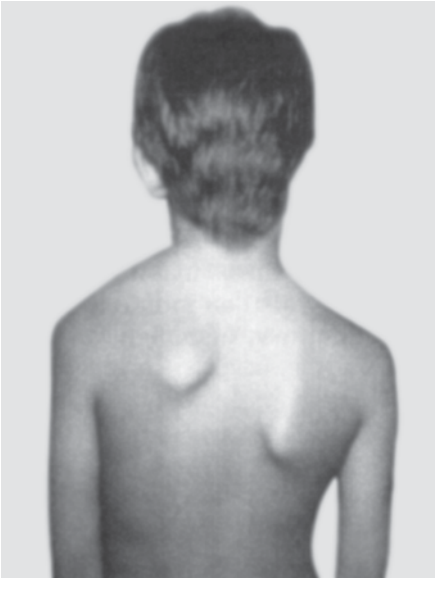


Fig. 10.25: Sprengel shoulder

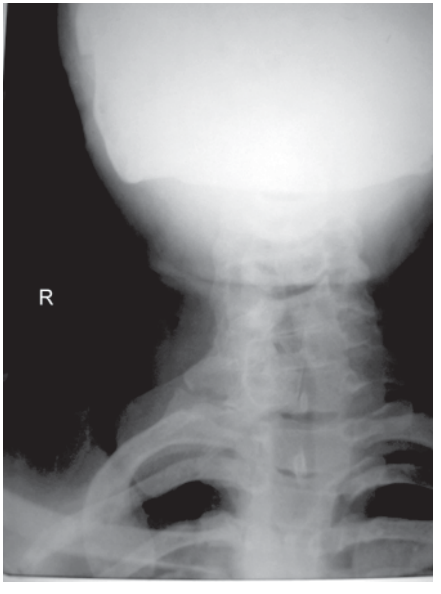


Fig. 10.26: Bony omovertebral bar on the right side with high riding scapula

posterior element of spine, aneurysmal bone cyst—expansile lytic bubbly appearance of spine body and tumors like Ewing's and osteosarcoma of vertebra. Painful left thoracic scoliosis can be associated with spinal dysraphysm—tethered cord or syringomyelia.

11 CHAPTER

Examination of Hip

The hip joint is the largest ball and socket joint with extreme degrees of movement in all directions. Its versatile movements are important for standing, walking, running, jumping, sitting cross-legged, sexual activities and childbirth. The examination should be in a systematic fashion. Introduce yourself and ask the name, age, and occupation.

PRESENTING HISTORY

Pain

Ask for onset, duration, nature of pain, aggravating and relieving factors—pain on activity or at rest, pain when first setting off to walk, pain that stops patient walking, i.e. start up pain may be different from stopping pain, i.e. loose femoral stem in failed THR or differential pain from spinal and vascular claudication; and radiation. Night pain indicates the severity of pain in arthritic conditions either due to osteoarthritis (OA), rheumatoid arthritis or infective arthritis (TB). Hip pain is mostly over the groin, buttocks area and sometimes radiates to the inner aspect of thigh up to the knee. Kids with hip problem present with referred pain in the knee, which is through the innervation of obturator nerve. Ask about numbness and pins and needles to identify neurological problem or spine pathology.

Level of Activity

This includes the walking distance, day-to-day activities, performance of job, hobbies including sport, etc. Each should be quantified to assess the disability and to plan treatment.

Stiffness

Patients explain this in their own terms like inability to bend forwards, inability to reach toenails to trim, difficulty in putting socks or shoes, inability to squat on Indian toilet and tricks patients employ to get dressed and do activities. Patient may also mention about morning stiffness in arthritic conditions.

Limp, Shortening or Deformity

Shortening and fixed deformities can lead to tilt of the pelvis and limp, which can be a presenting feature. Limping can also be due to pain or weakness of abductor mechanism.

Others

History of multiple joint problems especially other hip involvement (avascular necrosis, dysplasia of hip), constitutional symptoms, loss of weight and appetite (tuberculosis, secondaries in subtrochanteric area) and any history of trauma must be recorded.

Past History

Hip problems, significant medical problems like tuberculosis, diabetes, hypertension, childhood problems in hip-sepsis, obstetric history, if known, that is breech presentation, milestones and congenital anomalies; hip operations, steroid intake, occupational exposure (Caison's disease),

etc. Abdominal, urological and gynecological symptoms with referral to hip joint must be kept in mind and asked for.

Personal and Social History

Married or unmarried, smoking, alcohol (avascular necrosis), hobbies, the type of house (stairs) and toilet facilities.

Family History

Any similar problems.

Treatment History

Allergies, all types of treatment patient has had (tablets, local injections, physiotherapy, surgery) and the response to it.

What is Patient's Expectation?

Treatment is dictated by the needs of the patient, employment, that is, type of job being done now and possibilities in future, specific activities needing unusual or prolonged hip activity-religious activities.

GENERAL EXAMINATION

Local Examination

The sequence of examination in walking, standing, sitting and then lying down posture is ideal. It is cumbersome to ask the patient to change the examination position every time for our convenience. The economy of movements and smooth flow of examination gains confidence of the patient. It is better to go through look, feel and move at each step before you change the position of the patient.

Gait

Walking with and without walking aids.

This is analyzed with brisk walking on bare foot.

a. *Short limb gait:* Here the patient walks either with equinus of the ankle on the affected side to compensate for the short limb or one can notice drooping of the shoulder and pelvis towards the same side. When the patient stands he or she can compensate for the shortening by keeping the unaffected leg bent **(Fig. 11.1)**.
b. *Trendelenburg gait:* On weightbearing on the affected side, the weak abductors fail to raise the pelvis on the opposite side. So the center of gravity fails to shift towards the weight bearing leg. To achieve balance the upper trunk sways towards the weight bearing leg to move the center of gravity closer to weight bearing leg. If the Trendelenburg gait is present bilaterally it produces typical waddling gait (duck walking).
c. *Antalgic gait:* Patient has a short stance phase (less time of weightbearing on the affected leg) and short stride walk (small steps).
d. *Stiff hip gait:* Very little movement of the hip characteristically produces swinging of the pelvis with circumduction of the leg to clear the ground.

Walking aid and footwear assessment done.

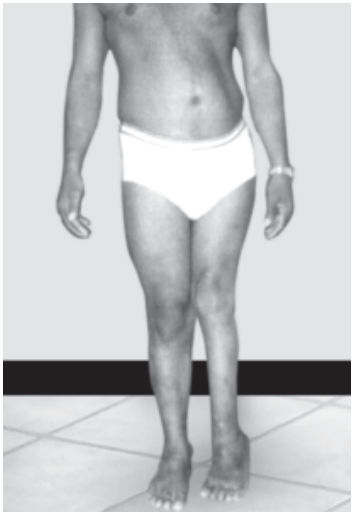


Fig. 11.1: Short left leg

PATIENT STANDING

Examination from Front

Assess the attitude and deformity in hip, knee and foot. Note any scars, skin changes, swelling, contours and muscle wasting especially quadriceps.

Assess shortening of leg by block test: With appropriate size blocks under the foot assess the shortening by achieving both anterosuperior iliac spines at same level.

Trendelenburg's test (Fig. 11.2): There are many ways to do this test and you must be comfortable with the way you do. From front feel for the anterosuperior iliac spine (ASIS) with your thumb and position yourself by kneeling down to the level of pelvis. The first bony prominence felt while palpating the groin from below and medial is anterosuperior iliac spine. Ask the patient to stand on sound leg lifting the other leg by flexing the knee but not the hip. The opposite side pelvis tilts up due to the abductor muscle function on the sound leg. Standing on the affected leg (single leg stance) causes drooping of the opposite side pelvis due to inefficiency of abductor mechanism. This can be well-appreciated by feeling the anterosuperior iliac spine. The positive test is when the opposite side anterosuperior iliac spine drops (sound side sags). This test was originally described by looking at the back of the patient exposing the buttocks and asking the patient to do single leg stance. The drop in the buttock fold (gluteal fold) must be appreciated on the non-weightbearing side by standing on the affected leg in a positive test.

Apley's method: Place one hand on shoulder of test side. Use the other hand to support the hand of opposite side. This hand is to assess the downward pressure. Now ask the patient to stand on test side. The other leg, which is lifted off the ground, should only be bent at the knee. The positive test is indicated by downward pressure exerted on supporting hand. This is a more elegant test and can be done without stripping the patient in the outpatient clinic **(Figs 11.2 and 11.3A and B)**.

I prefer to do Trendelenburg's test by combining the ways described above. Kneeling in front of the patient, feel the anterosuperior iliac spines and place the patient's hands on either side of examiner's shoulder. Now ask the patient to stand on the affected leg. Positive Trendelenburg's test is indicated by downward pressure exerted by the patient on the examiners shoulder with the opposite hand and also the sagging of the opposite anterosuperior iliac spine. This method confirms both by tactile perception and traditional way of assessment.

The single leg stance should be maintained for at least 30 seconds as delayed Trendelenburg's test could be positive. False-positive test can occur with fixed abduction contracture, painful abduction, in poor balance due to generalized weakness especially in elderly people. The test can be positive with any disorder affecting the fulcrum (hip), the lever arm (neck) and the power (abductor muscles).

Examination from Side

Assess for flexion deformity at hip, knee and exaggerated lumbar lordosis.

Examination from Back

Look for scars, wasting of gluteal muscles, Trendelenberg test noting the buttock-fold raise or drop.

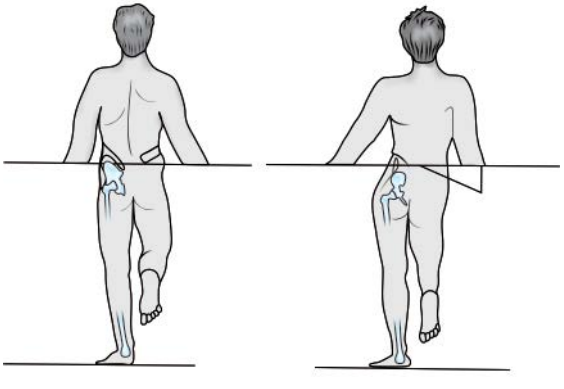
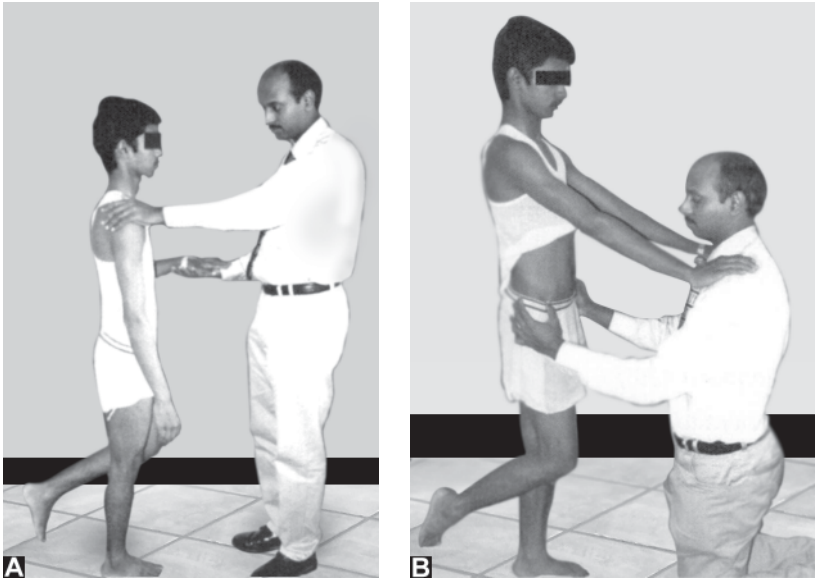


Fig. 11.2: Trendelenburg's test



Figs 11.3A and B: Trendelenburg's test-alternate method

SUPINE ON COUCH

Look

Reconfirm all the inspection findings like exaggerated lumbar lordosis, attitude of the leg, deformity, wasting, leg length discrepancy, etc.

Feel

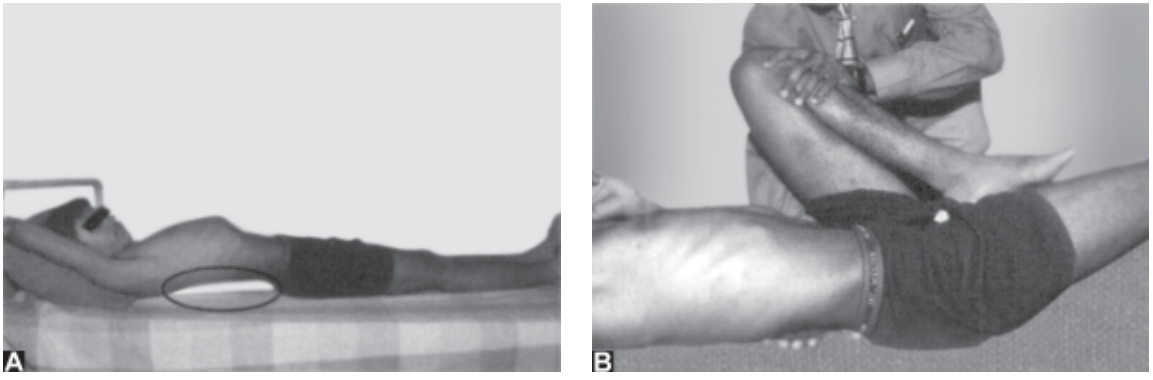
Ask the patient for tender spot and feel this last. Always look at the patient's face. Feel for tenderness along greater trochanter (trochanteric bursitis), anterior joint line, that is along the groin, and feel for lymphadenopathy. Feel for any abnormal bony mass. Femoral pulse can be feeble in developmental dislocation of hip (Vascular sign of Narath).

Move

Flexion (Normal 0-120°)

Ask the patient to actively flex to have an idea of pain-free movements. The primary flexors are iliacus and psoas muscle and accessory muscles of help are rectus femoris, sartorius, tensor fascia lata, pectineus, and adductors. Strength of iliopsoas is tested by placing the patient in sitting posture to fix the pelvis and actively ask the patient to elevate the flexed knee against resistance. To assess fixed flexion deformity (FFD) Thomas test is done. Place hand behind the back to assess lumbar lordosis. The examiner flexes the patient's normal hip and knee until the lumbar lordosis is obliterated. Now the affected hip reveals the fixed flexion deformity if any. Ask the patient to clutch the unaffected bent knee to the chest and flex the affected hip from the position of fixed flexion deformity to full further flexion to assess the range of movement (e.g. FFD: 30°; ROM: 30°-110°) (**Figs 11.4A and B)**.

If both hips are affected flex both hips to eliminate lumbar lordosis and then gently extend one hip passively. Stop when the lumbar lordosis begins to reappear. This is the fixed flexion deformity of that hip. Similarly repeat the test for the other hip.



Figs 11.4A and B: Thomas test

In special cases where there is fixed flexion deformity of knee, place the patient so that the knee is at the edge of the couch to eliminate the effect of FFD at knee.

In patients with deformed femoral head or any rotational deformity, on bending the hip an 'axis deviation' can be appreciated.

Example: Normally on flexing the hip the knee approaches the same shoulder, but in slipped capital femoral epiphysis with fixed external rotation deformity there is axis deviation on flexing, with knee pointing out on extreme flexion.

Rotation in Extension

In extended knee rotate the leg by holding the foot with one hand and the other hand over the lower thigh and turn the foot in and out for internal and external rotation, respectively **(Fig. 11.5)**.

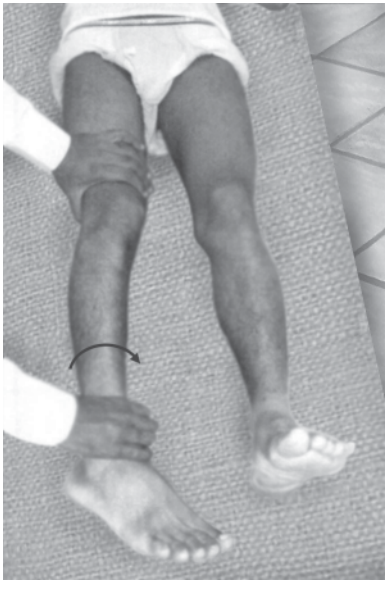


Fig. 11.5: Testing internal rotation of hip with knee extended

Rotation in Flexion

Hip flexed to 90°, knee flexed to 90° use leg as a lever arm to do internal rotation (Normal 0 to 45°) by turning the foot out and external rotation (Normal 0 to 45°) by turning the foot in. This test can be done in prone position especially in children for more accuracy. Differential range of rotational movements in flexion and extension positions of the hip indicates deformed femoral head (Perthes' disease, avascular necrosis of hip).

The prime internal rotators are gluteus minimus and tensor fascia lata. Accessory muscles that help in internal rotation are gluteus medius, semitendinosus and semimembranosus. The prime external rotators are obturator externus and internus, piriformis, superior and inferior gemelli, gluteus maximus and sartorius **(Figs 11.6 and 11.7)**.

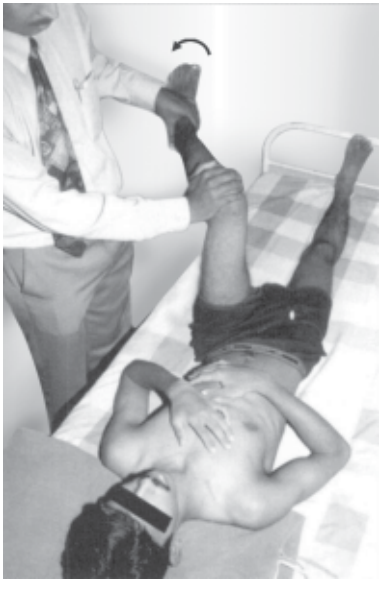


Fig. 11.6: Testing internal rotation of hip with knee flexed

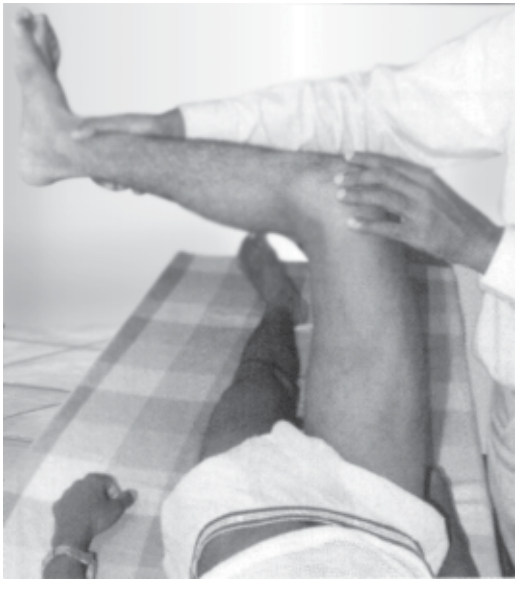


Fig. 11.7: Testing external rotation of hip with knee flexed

Abduction/Adduction in Extension

Try active movements first. Active adduction is done by adductor magnus, brevis, longus, pectineus and gracilis. Active abduction is primarily done by gluteus medius and accessory muscles of help are gluteus minimus, tensor fascia latae, upper fibers of gluteus maximus. Keep the hip and knee extended, fix the pelvis after squaring the pelvis as mentioned below. The pelvis can be fixed in children by palpating both anterior superior iliac spines (ASIS) with one hand span. In adults the pelvis can be fixed with forearm and the other hand used to move the leg. Now assess the range of movements of abduction (Normal 0 to 40°) and adduction (Normal 0 to 25°) **(Figs 11.8 and 11.9)**.

Abduction in Flexion

In neonates and young children it is easy to appreciate the limitation of abduction with hips and knees flexed, and performing simultaneously on both sides. Abduction in flexion is easy because of the flexion attitude of the limbs in young kids. Normally the thigh should touch the couch on abduction.

Telescoping test: Fix the pelvis with one hand, thumb feeling the ASIS and forefinger on greater trochanter, flexing the hip and knee 90°. Hold the leg with other hand and give axial force along the thigh and feel for superior displacement of trochanter in relation to ASIS **(Fig. 11.10)**.

Patrick's 'faber' test (flexion, abduction and external rotation test): This produces click and pain in osteoarthritis or in acetabular labral tear **(Fig. 11.11)**.

Measurement

Apparent leg length discrepancy can be measured from xiphisternum to medial malleolus keeping the body and legs parallel to the couch and not making any attempt to square the pelvis. This gives the functional leg length discrepancy **(Fig. 11.12)**.

Feel for anterior superior iliac spine and make pelvis square (line joining both ASIS is perpendicular to the couch). Attempt to make

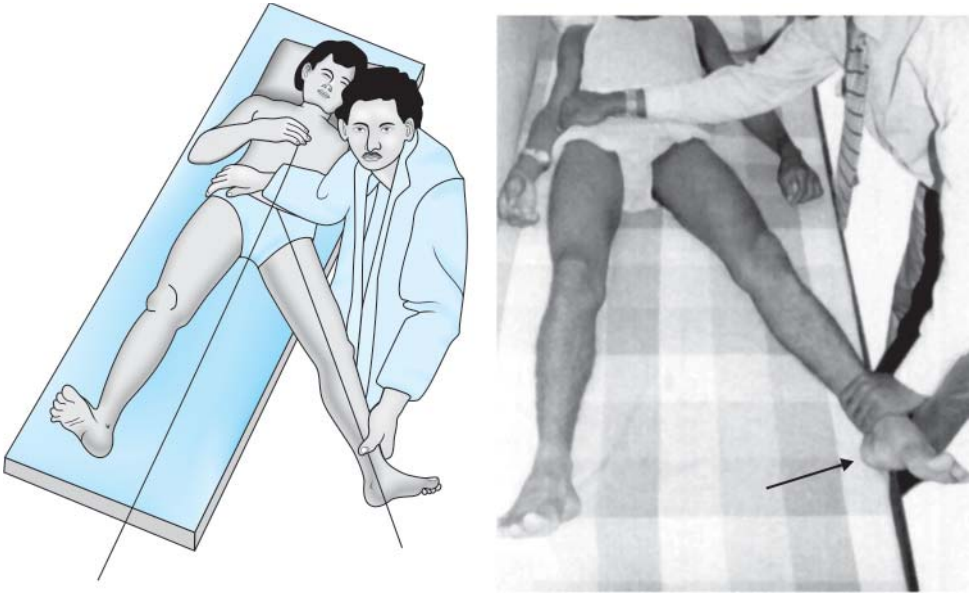


Fig. 11.8: Testing abduction of hip with knee extended

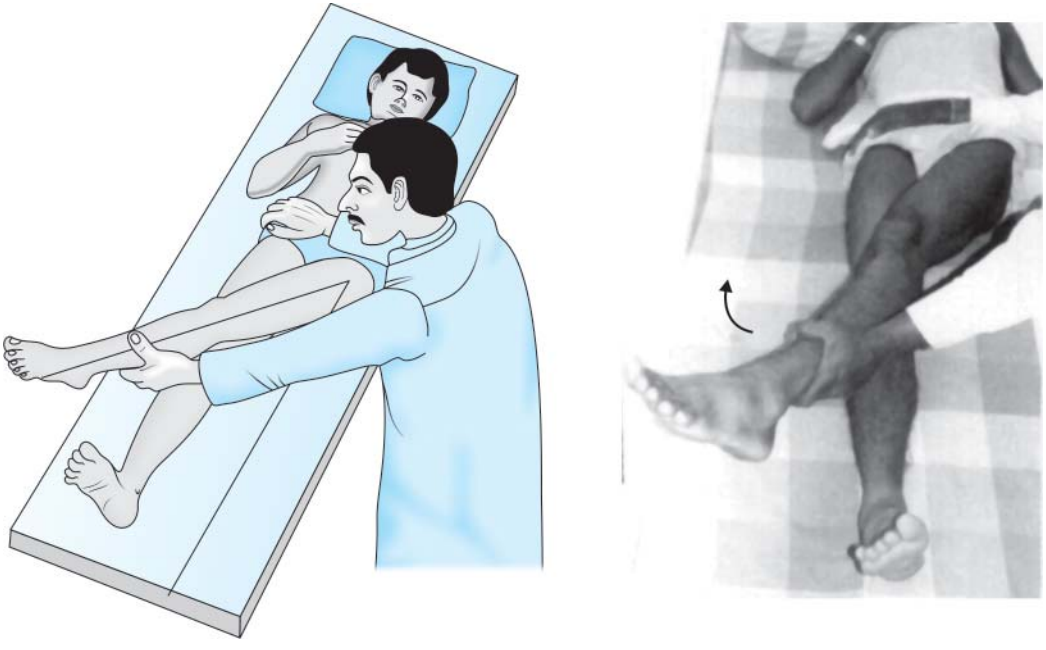


Fig. 11.9: Testing adduction of hip with knee extended

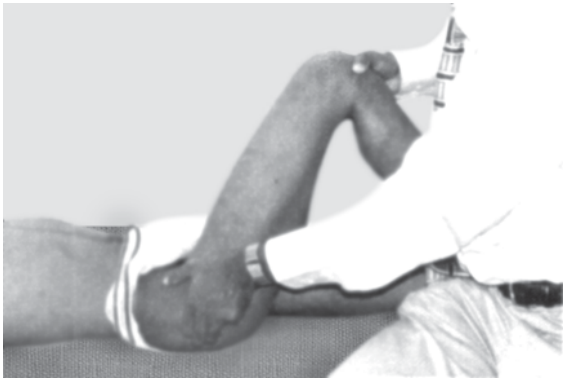


Fig. 11.10: Telescoping test

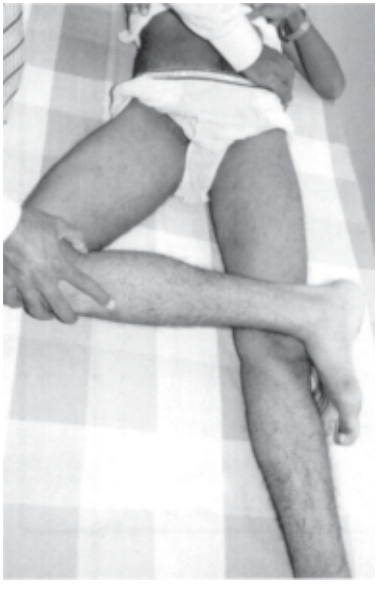


Fig. 11.11: Faber test

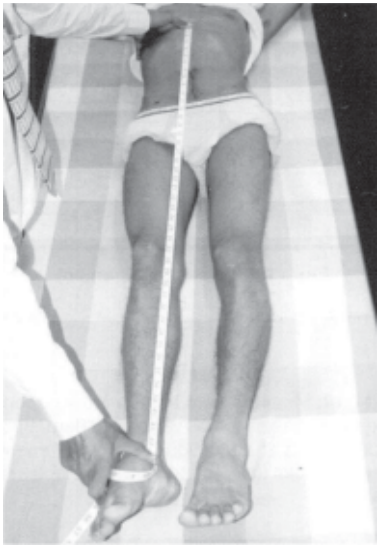


Fig. 11.12: Apparent measurement

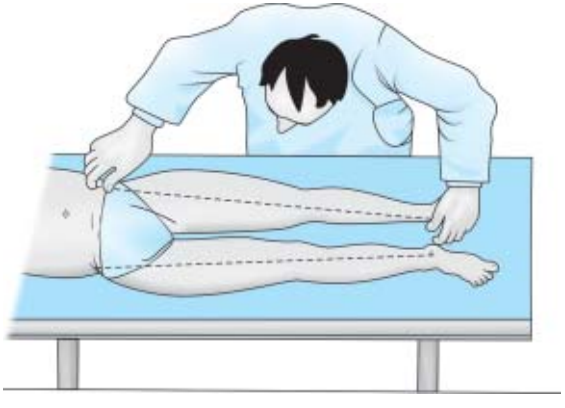


Fig. 11.13: True measurement

legs parallel to the couch with pelvis squared. Now measure the leg length from ASIS to medial malleolus. This is the true length measurement. Make sure the legs are in identical position and attitude **(Fig. 11.13)**.

In abduction contracture the ASIS will be at a lower level on the affected side when the legs are parallel and to square the pelvis the leg needs to be abducted. The point when the abduction makes the pelvis square indicates the degree of abduction contracture from the neutral position. Further free abduction is assessed from this point (e.g. fixed abduction deformity 20 degrees; free abduction 20 to 40 degrees).

In adduction contracture the ASIS will be at a higher level on the affected side when the legs are parallel. Adducting the affected leg further until both ASIS are perpendicular to the table can square

the pelvis. The degree of adduction necessary to square the pelvis is the fixed adduction deformity. Further free adduction is assessed from this point (e.g. fixed adduction deformity 20 degrees; free adduction 20 to 40 degrees).

In both adduction and abduction contracture true leg length measurement can be done only after squaring the pelvis and measuring each leg in the same identical position **(Fig. 11.14)**. Please note, the adducted leg crosses over the other leg and is measured from ASIS to medial malleolus. The other leg needs to be crossed in the same way, maintaining the same degree of adduction before measuring from ASIS to medial malleolus.

Flexion, varus or valgus deformity of the knee will affect the measurement of the legs. In such cases it is better to measure thigh and leg segments separately. ASIS to knee joint line or tibial tuberosity, and from there to medial malleolus.

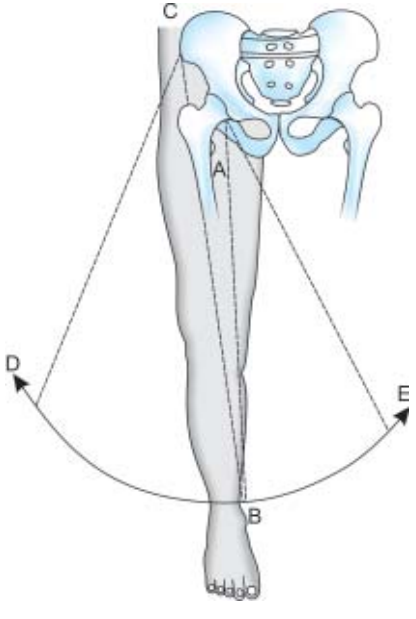


Fig. 11.14: Measurement from anterosuperior iliac spine to medial malleolus varies in different positions of adduction and abduction but measurement taken from the hip joint (fulcrum) to medial malleolus remains constant

Galleazi's or Allis's Sign

Flex knees to 90° with hips and ankles at 45°. Keep both malleoli at same level. Note the level of knees and parallelism of femora and tibia. If knees are at different levels, and tibia are parallel, the discrepancy is in tibia. If femora are parallel the discrepancy is in femur **(Fig. 11.15)**.

Bryant's Triangle

Identify ASIS with thumb and tip of greater trochanter with forefingers. Assess the perpendicular distance between the two points. This gives an idea on supratrochanteric shortening. Supratrochanteric shortening can be due to fracture neck of femur, traumatic dislocation of hip, developmental dislocation of hip, avascular necrosis with collapse, arthritis hip—rheumatoid, infective and coxa vara.

Roser-Nelaton's line: If both hips are affected construct Roser-Nelaton's line. This is a line joining ASIS to ischial tuberosity. The tip of the greater trochanter should touch this line normally **(Fig. 11.16)**. If it lies above this line supratrochanteric shortening is confirmed.

Chiene's lines: The lines joining the two ASIS and two greater trochanter are normally parallel to each other. This is disturbed if trochanter is shifted up **(Fig. 11.17)**.

Schoemaker's line: The lines joining the greater trochanter and ASIS when extended above from both sides may cross above the umbilicus in the midline normally. In supratrochanteric shortening on one side the lines may cross above the umbilicus away from the midline. In bilateral supratrochanteric shortening these lines may cross in the midline below the umbilicus **(Fig. 11.17)**.

Morris bitrochanteric line: The distance from pubic symphysis to greater trochanter is measured. This again indicates supratrochanteric shortening **(Fig. 11.17)**.

Infratrochanteric shortening is accounted by actual measurements of individual components

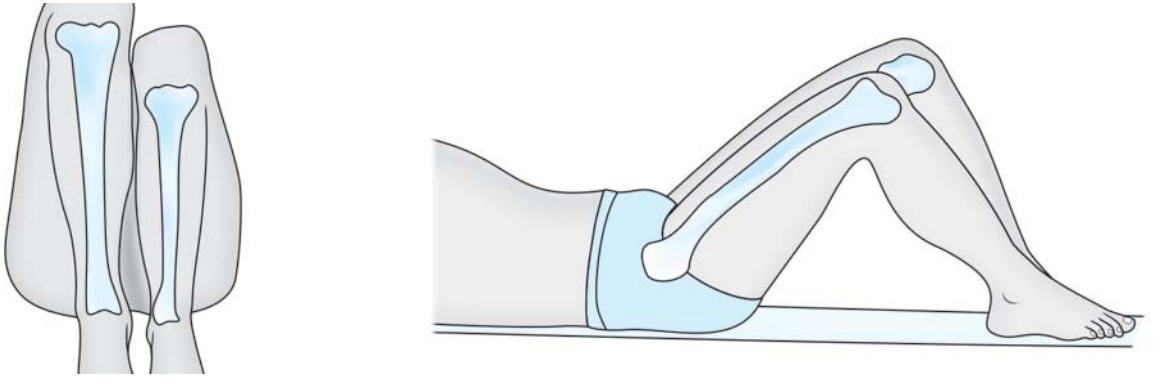


Fig. 11.15: Galleazi's sign

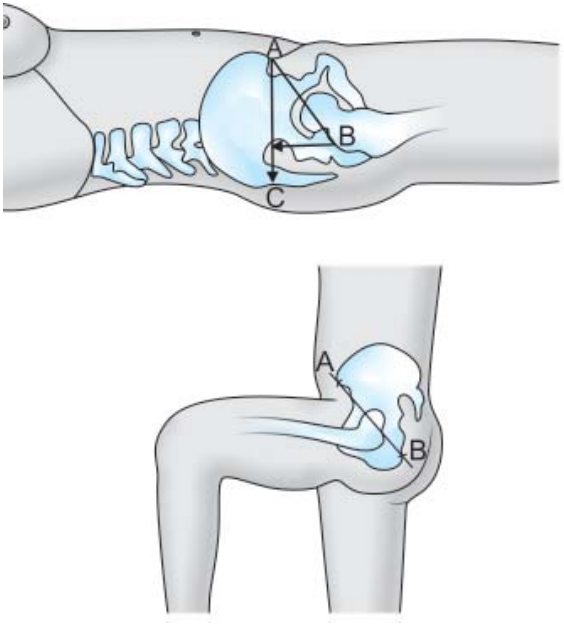


Fig. 11.16: Bryant's triangle and Nelaton's line

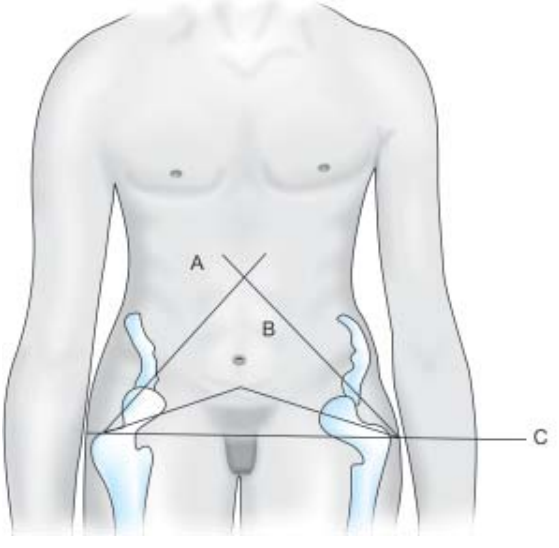


Fig. 11.17: Showing the construction of A: Schoemaker's line, B: Chiene's line, C: Morris bitrochanteric line

of femur and tibia. This can be from femoral shaft or tibia or calcaneum malunited fracture or from growth disturbance.

LATERAL ON COUCH

Test active abduction and also palpate the abductors for power against resistance. Draw Roser-Nelaton's line if necessary.

PRONE EXAMINATION

Examine the spine, buttocks for any scars, wasting of muscles, extension (Normal 0 to 15°), internal and external rotation of hip **(Figs 11.18 to 11.20)**. Gluteus maximus is the prime mover of extension of hip with the accessory muscles of help are semimembranosus, semitendinosus and biceps femoris. Feel for

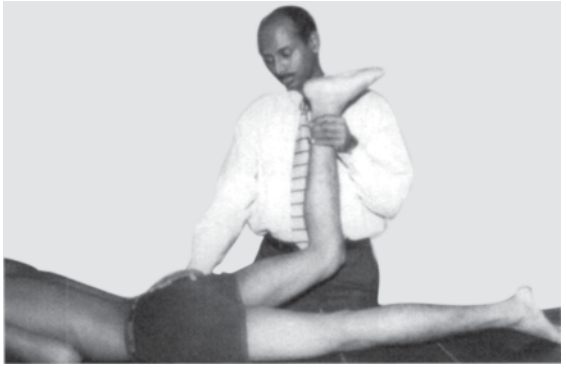


Fig. 11.18: Testing extension of hip

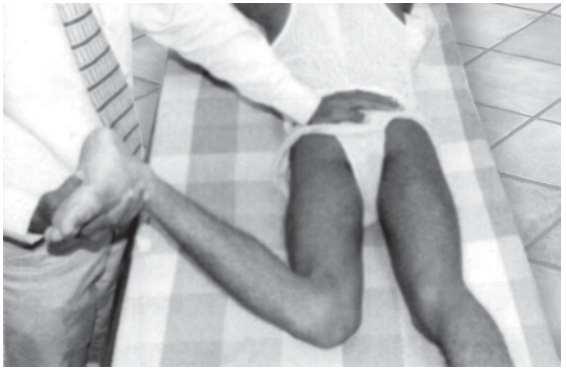


Fig. 11.19: Testing internal rotation of hip with knee flexed in prone position

any abnormal mass (dislocated femoral head or myositis ossificans).

Prone examination is useful to assess the version (anteversion or retroversion) of femur as discussed in the assessment of rotational malalignment of the leg (Chapter 14).

Prone on couch is avoided if patient cannot lie because of pain or severe fixed flexion deformity of the hip or obesity or other medical problems.

In children with bilateral fixed flexion deformity, flexion deformity of each hip can be revealed in prone examination. Bring the patient's pelvis to the edge of the couch so that the thighs hang down and can be flexed. Now

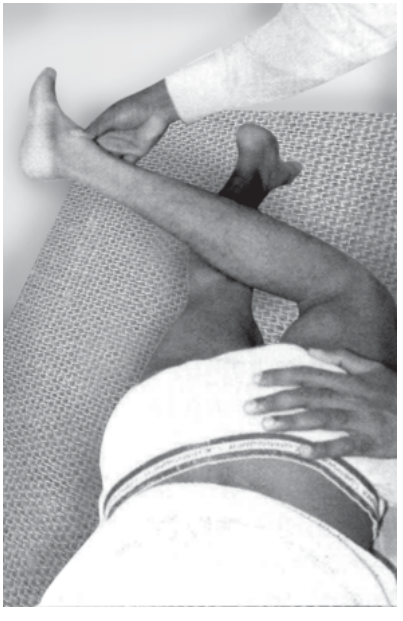


Fig. 11.20: Testing external rotation of hip with knee flexed in prone position

bend both hips to obliterate the lumbar lordosis, that is, the lumbar spine is flat and parallel to the couch. Extend one hip at a time and check when the lordosis begins to reappear. This indicates the FFD of that hip. Repeat the same in the other hip. The examiner may need an assistant to hold the legs that hangs free from the couch.

Examination is completed by sacroiliac stress test, opposite hip examination, examination of ipsilateral knee and foot. Spine should be examined including neurological assessment to rule out any radiating pain.

General abdominal examination—femoral hernia, urological problem and other pathology giving hip pain vascular supply (saddle thrombus—buttock and leg pain) must be considered.

CONDITIONS AFFECTING HIP

Developmental Dysplasia of Hip (DDH)

History: Birth history—breech presentation, first born female child, developmental milestone,

family history of DDH. Mother may notice difficulty in abduction while putting nappy or can present late at the age of walking with limp or short leg.

Clinical Signs

The clinical signs to be assessed are:

Barlow's test: The baby must be relaxed and it is helpful to examine the baby shortly after a feed. Hold both the lower extremities in such a way that the knees are flexed to 90 degrees and the upper thigh is held with the thumb along the medial aspect and the middle or ring finger behind the greater trochanter.

The hip is adducted while pressing down gently on the knee with the palm and inside of the thumb. An unstable hip may dislocate with a soft cluck. This is a test for dislocatable hip **(Fig. 11.21)**.

Ortolani's test: The child is held as above; the hip is then flexed to 90° and gently abducted while lifting the greater trochanter upwards with the fingers. A dislocated hip may reduce with a clunk. This is a test for dislocated hip.

Telescoping test: The pelvis is fixed, the thigh is grasped above the knee and the hip is flexed to 90°. The thigh is pushed and pulled along the axis of the femur while the other hand feels the relationship between the greater trochanter and anterosuperior iliac crest. Test is positive in free proximal and distal movement of greater trochanter in DDH, old unreduced posterior hip dislocation, dislocated total hip replacement, fracture neck of femur or loss of head/neck.

Trendelenburg's test: This test is done in an older child who can understand and cooperate. This test is done as described earlier.

Galleazi's sign or Allis's sign: Shortening of the limb from pelvis to knee is observed by flexing the knees and the hips as described before.

Asymmetrical Skin Crease

Restricted abduction Foot anomalies, torticollis, plagiocephaly and associated congenital anomalies.

X-ray finding is illustrated in **Figure 11.22**.

Osteoarthritis (OA)

It is a degenerative wear and tear process of hip joint of primary or secondary etiology. Primary OA is common in elderly population while secondary OA can occur at any age.

Predisposing factors are obesity, trauma-fracture or dislocation, AVN, dysplasia, post-infective sequelae, etc.

Fig. 11.21: Testing for DDH, Barlow's test

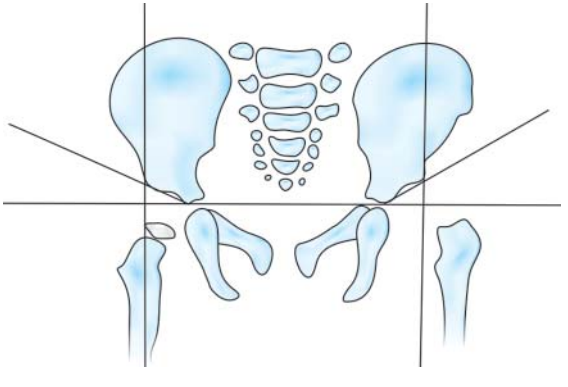


Fig. 11.22: Pelvis lines-horizontal Hilgenreiner's line is through triradiate cartilage and vertical Perkin's line is through the outer rim of acetabulum. Normally the capital femoral epiphysis is in inner and lower quadrant

Patient presents with pain over the groin or buttocks area, morning stiffness, inability to trim toe nails or tie shoe lace, difficulty to squat, limp, decreased walking distance and restricted movements. Clinical examination reveals antalgic gait, limitation of movements especially internal rotation, fixed deformities and rarely shortening of leg (shortening is common in secondary OA).

Radiologically OA of hip can be medial, lateral or global arthritis.

Findings are:

a. Narrowing of joint space
b. Marginal osteophytes
c. Subchondral sclerosis (overweight bearing part of acetabulam)
d. Subchondral cyst formation (acetabular or femoral head side)
e. Signs of causes of secondary OA.

Inflammatory Arthritis

This is of two types-rheumatoid and non-rheumatoid.

Patient presents with pain, stiffness, deformity and limitation of activities of daily living associated with other joint problems.

Non-rheumatoid arthropathy commonly affects hips (ankylosing spondylitis, psoriatic arthritis, etc.) usually results in progressive stiffness and sometimes ankylosis of joint.

Rheumatoid arthritis commonly affects small joints but can affect hips to produce laxity of the joint with destruction of femoral head and sometimes protrusio acetabuli. The other hip involvement is common.

Avascular Necrosis (AVN)

It is a rare condition affecting the hip joint in the age group 20 to 50 years. Fifty percent is bilateral and in steroid induced AVN it is up to 80 percent bilateral.

Mostly idiopathic; secondary causes include-trauma (fracture or dislocation), drugs (steroids, antiepileptics, chemotherapy, indomethacin), alcohol, Gaucher's disease, dysbaric osteonecrosis, irradiation, etc. Young patient presenting with hip pain and limitation of movements especially internal rotation must raise suspicion of AVN.

X-ray may be normal in the initial stage, later on subchondral fracture, deformation of head, sclerosis, cyst formation and secondary OA changes may occur. MRI scan is diagnostic at early stage.

Legg-Calve-Perthes Disease

Idiopathic necrosis of capital femoral epiphysis can cause collapse, fragmentation and deformity of the femoral head. It affects boys four times more commonly than girls. It usually affects only one hip and bilateral involvement is 10 to 15 percent. Typically presents between the ages of 4 to 8 years, short stature and delayed milestones (small for age) has been associated with the disease.

Patient may present with insidious onset groin or anterior thigh pain or knee pain and limp. The limb is held in flexion, adduction and external rotation attitude. Limitation of abduction and internal rotation are early signs of hip irritation.

Differential diagnosis

- Unilateral—septic arthritis, transient synovitis of hip and epiphyseal dysplasia.
- Bilateral—multiple epiphyseal dysplasia, hypothyroidism.

Slipped Upper Femoral Epiphysis (SUFE)

It is a cause for pain in boys of 12 to 14 years and girls of 10 to 12 years age group. Commonly unilateral and bilateral in 30 percent of cases. Obesity, rapid growth spurt and endocrinopathy like hypothyroidism and renal rickets can predispose to slip. This condition is a consequence of an imbalance between the forces that stabilize the epiphysis and the normal mechanical forces that may tend to displace it.

The presentation can be acute slip (<3 weeks), acute pain presenting after trauma or in chronic slip insidious onset of chronic pain in the groin or anterior thigh or medial aspect of the knee. Acute on chronic slip can also be a presentation. Patient can have FFD

due to muscle spasm (paradoxical to extension deformity at the site of slip) and external rotation deformity of the leg. Limitation of abduction due to varus deformity can also be demonstrated. Shortening of leg may be seen in severe slip.

X-ray anteroposterior and frog leg lateral is recommended.

Widening of physis may be the subtle early sign on comparison with opposite normal hip, with decrease in the height of epiphysis.

Metaphyseal blanch sign, a dense area seen in proximal metaphysis, which may be due to healing process or superimposition of posteriorly rotated head on metaphysis.

Capener's sign—A line (Kliene's line) drawn along the superior border of the femoral neck passes through the superior corner of the epiphysis normally but will not do so after a slip.

Lateral view shows the slip clearly.

Chondrolysis and avascular necrosis are recognized complications of SUFE.

Tuberculosis of Hip

It is the second most common bony site of tuberculous infection after the spine. The infection starts in the acetabulam or the head of femur (Babcock's triangle).

Limp is the earliest complaint, initially after walking and later even after rest. Patient may have pain referred to the thigh or knee and night cry. Muscle wasting is present. Thomas' test will reveal the fixed flexion deformity of hip and rotations will be restricted.

Deformities are seen depending on the stage of hip involvement.

Stage 1 (Stage of synovitis)—effusion causes flexion, abduction and external rotation of hip with apparent lengthening of the limb.

Stage 2 (Stage of arthritis)—muscle spasm causes flexion, adduction and internal rotation of hip with apparent shortening of the limb.

Stage 3 (Stage of erosion)—destruction of the joint and dislocation of the femoral head causes true shortening of the limb.

Transient Synovitis of Hip

It is a common self-limiting condition of unknown etiology where there is nonspecific inflammation of the joint in children less than 8 years old. It can be a post-viral reaction. The child usually presents with sudden onset limp and pain with no history of injury. It must be differentiated from septic arthritis by normal well-being of the child (not irritable, no septic focus and no fever) and normal blood tests (full blood count and ESR).

Acute Septic Arthritis Hip

Common in neonates and children with classical presentation of pseudoparalysis of the lower limb due to pain. Child is very reluctant to move the leg and will not put weight on that leg. Spasm and pain with gross restriction of hip movements are the features. Child will be sick, irritable and feverish with classical attitude of position of ease, of flexion, abduction and external rotation. Blood test, Ultrasound scan and aspiration/culture, and MRI scan can confirm the diagnosis.

Trochanteric Bursitis

Patients present with localized pain, tenderness and swelling over the greater trochanter area. It is due to irritation of overlying soft tissue (iliotibial band) due to constant friction in flexion-extension movement. It is common in sports person, following lateral hip surgery, trochanteric internal fixations and rarely infection, but most of the time it is idiopathic in origin. Pain is aggravated in flexion adduction movement or on sitting cross-legged.

Femoroacetabular Impingement

It is a recognized cause of pain and early arthrosis in young adult hip. Ganz described two types: CAM type presenting more commonly in young males and pincer type more commonly in women of late 30s or early 40s. Cam type impingement is defined as insufficient offset or concavity of femoral head neck junction causing shearing

damage to the labral-chondral junction and acetabular articular cartilage. In pincer-type impingement the primary deformity is on acetabular side in the form of overcoverage (protrusion, coxa profunda or acetabular retroversion) leading to abutment of femoral neck against acetabular rim, pinching the labrum between the femoral neck and the bony rim. It also typically causes a thin rim of acetabular articular damage. Young people present with insidious onset of hip pain related to activity and in certain cases related to specific injury. Pain is typically in midgroin or referred to buttock area often localized by the patient by grasping the hip between thumb and index finger, the so-called 'C' sign. Exacerbation of pain after long periods of sitting or sporting activity with periods of rest from their activity providing some relief and physical examination reveals limitation of internal rotation in flexion. Flexion, adduction and internal rotation can reproduce their pain with positive impingement test.

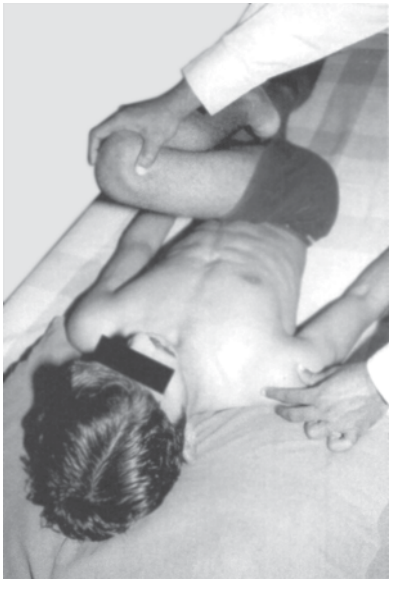
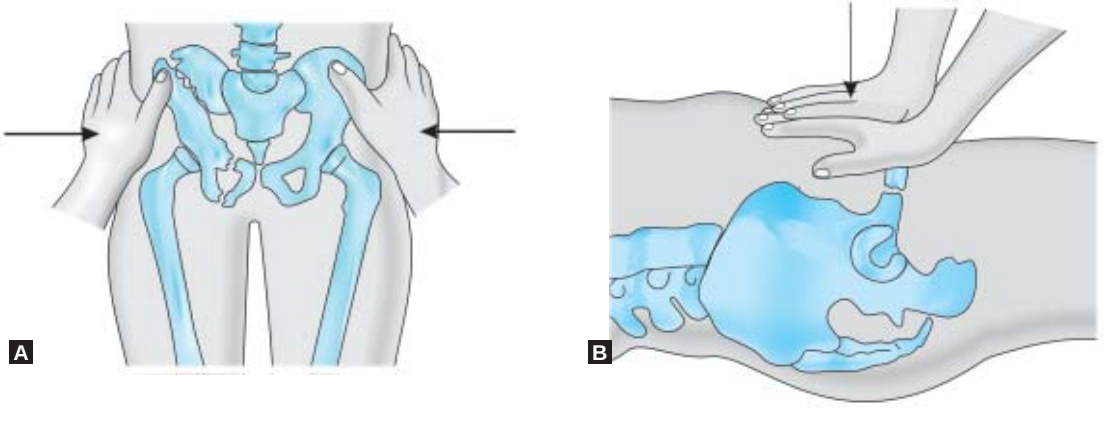


Fig. 11.23: Pump handle test

SACROILIAC JOINT (SIJ) STRESS TEST

Sacroiliac joint becomes diseased in inflammatory conditions (seronegative inflammatory arthropathy), infections and fracture-dislocation. Special stress tests and other diagnostic tools can identify the pathology. Turning on bed especially in early morning hours and getting up from bed is very painful. Sometimes there can be pain on weightbearing with hamstring spasm.



Figs 11.24A and B: (A) Lateral and (B) anterior to posterior compression is applied to provoke pain in the supine patient with pelvic fracture

Genslen's test: The hip and knee of the affected side are flexed to fix the pelvis and the hip on the unaffected side is hyperextended over the edge of the examination table. This will exert rotational strain on the SIJ.

Pump-handle test: Patient supine, grasp the limb just below the knee and steady the trunk by grasping the shoulder on the same side. Fully flex the hip and knee joints and direct the flexed knee steadily towards the opposite shoulder. The test is positive if pain is experienced in the SIJ **(Fig. 11.23)**.

Faber test: The lower limb is forced into flexion, abduction and external rotation at the hip. This causes pain at SIJ.

Compression and distraction stress test: Forceful compression over both iliac crests inwards or forceful distraction of pelvis over both ASIS outwards can cause pain in sacroiliac disruptions or in pelvic fractures **(Figs 11.24A and B)**.

12 CHAPTER

Examination of Knee

Knee joint is the largest joint of the body with poor inherent bony stability and depends on the ligaments and muscles for static and dynamic stability. A systematic examination can help the examiner to identify and accurately diagnose a knee problem. Introduce yourself to the patient; ask his/her name, age and occupation.

PRESENTING COMPLAINT

Pain

Onset, duration, location of pain and point of maximum pain should be asked for. Knee pain can be a referred pain from hip pathology especially in children. Pain limiting the level of activities should be assessed by the walking distance, work and hobbies. Pain on climbing stairs or coming down stairs indicates quadriceps or patellar mechanical problem. Catching pain on turning movements indicates some mechanical pain due to chondrosis or meniscal injury or patellar malalignment or loose body. Clicking associated with pain in the front of the knee is mostly from patellofemoral malalignment. Tense effusion in the knee causes more pain. In severe injury with fracture there is less hemarthrosis due to capsular disruption and extravasations of blood, causing less pain. Patients with fixed flexion deformity of the knee can present with thigh pain. This is due to muscle fatigue from constant action of quadriceps in standing and walking posture. Normally on standing the knee joint gets locked in full extension by screw home movement (during terminal extension, femur internally rotates on tibia to lock the knee) and the quadriceps are at rest with no exertion. Constant pain and night pain may indicate infection or tumors or severe arthritis.

Swelling

Onset, duration, site of first appearance, change in size and shape, appearance in different positions of knee and associated pain must be recorded. A horseshoe shaped swelling around the patella and suprapatellar area is usually joint effusion. Time of appearance after injury is important. If it appears within an hour or two after injury it is most likely a hemarthrosis (anterior cruciate ligament [ACL] or posterior cruciate ligament [PCL] injury, intra-articular or osteochondral fractures, peripheral meniscus tear or tear in deep portion of joint capsule). Swelling which appears after 6 hours or next day is a sympathetic reactionary effusion. A localized swelling along the joint line can be due to meniscal cyst, ganglia or bursa.

Stiffness

This can be morning stiffness or inability to bend or straighten the knee fully.

Mechanism of Injury

It gives a clue to the diagnosis. A twisting injury with the foot resting on the ground can give rise to collateral ligament injury, meniscus tear and cruciate ligament rupture. Sudden deceleration can result in anterior cruciate ligament rupture.

In sports injury it is important to know whether the patient was able to walk or complete the game after the injury. A dashboard injury to the front of the knee can produce posterior cruciate ligament injury.

Giving Way

Sudden giving way of the knee can be due to quadriceps wasting or quadriceps inhibition from pain or swelling, or it can be due to ACL injury or combined ligamentous injury. Sometimes patellofemoral malalignment resulting in subluxation or dislocation can present with giving way of the knee. Patient walking in a straight line has no problem but sudden turning or twisting of the body leads to buckling with collapse and pain in the knee. This true giving way symptom is common in ACL tear or patellar instability. This flexion rotation injury is common in sports like foot ball and one should always enquire about whether the player was able to complete the game or discontinued.

Locking

Patient walking in a straight line has no problem but sudden turning or twisting of the body leads to buckling with collapse and pain in the knee. This knee buckling is common in patients with anterior knee pain with true giving way symptoms in ACL rupture and patella dislocation or subluxation. True mechanical locking results from meniscal injury where the patient is not able to straighten the leg from a fixed angle of flexion or there is sudden loss of terminal extension.

Pseudo-locking is what patient describes as a transient sensation of tightness and not being able to straighten or bend the knee. This can be from loose bodies or chondral flap tears or patellar subluxation. Pseudo-locking due to loose body happens at different degrees of knee flexion at different times. Patient sometimes describes that the locking corrects by maneuvering the leg in some position. The loose body felt by the patient in different sites must be noted—"joint mouse" **(Fig. 12.1)**.

Clicks

Most common in young adults due to patellofemoral disorders. Bilateral clicks near terminal extension can be due to discoid meniscus (meniscus is like a full disk covering the tibial condyle instead of a normal 'C'-shaped structure).

Miscellaneous

Other joint involvement, history of urethritis, conjunctivitis, bowel disturbances (inflammatory diseases, crystal arthropathy) and constitutional symptoms must be recorded.

Other relevant history must be asked for as discussed in Chapter 1.

What is Patient's Expectation?

General Examination

LOCAL EXAMINATION

Examine the patient—walking, standing, supine on couch and prone on couch.

Explain to the patient at each step what you are doing.

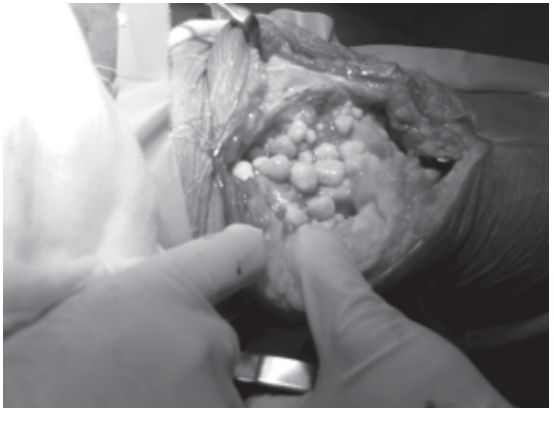


Fig. 12.1: Loose bodies in knee due to synovial osteochondromatosis *(For color version, see Plate 6)*

Look

Gait

Ask the patient to take off the footwear and to walk briskly to and fro. Comment on any walking aid or external appliances used.

Types of gait: Antalgic, lateral thrust, stiff knee, quadriceps gait (hand to knee gait in poliomyelitis patients with weak quadriceps where the hand is used to push the thigh back to lock the knee, or can internally rotate the leg to use iliotibial band as the extensor of knee).

Patient Standing

Looking from Front

Assess the knee alignment for deformity, varus (bow leg) or valgus (knock knee) deformity **(Figs 12.2A to C)**, patellar rotation (squinting patella to one side, usually laterally), foot rotation, any scars (note for tiny arthroscopic scars), wasting of quadriceps, swelling around the knee—diffuse horse-shoe shaped swelling around the patella due to fullness of suprapatellar pouch with obliteration of hollowness on either sides of ligamentum patella indicates effusion of the joint. Localized swelling around the knee can be due to ganglion, meniscal cyst or tumors.

Looking from Side

Ask the patient to push the knee back and assess flexion deformity or recurvatum of the knee. By looking from front and side triple deformity of the knee- flexion, posterior subluxation and external rotation of tibia can be made out (rheumatoid arthritis, tuberculosis). Look for any lateral swellings.

Looking from Behind

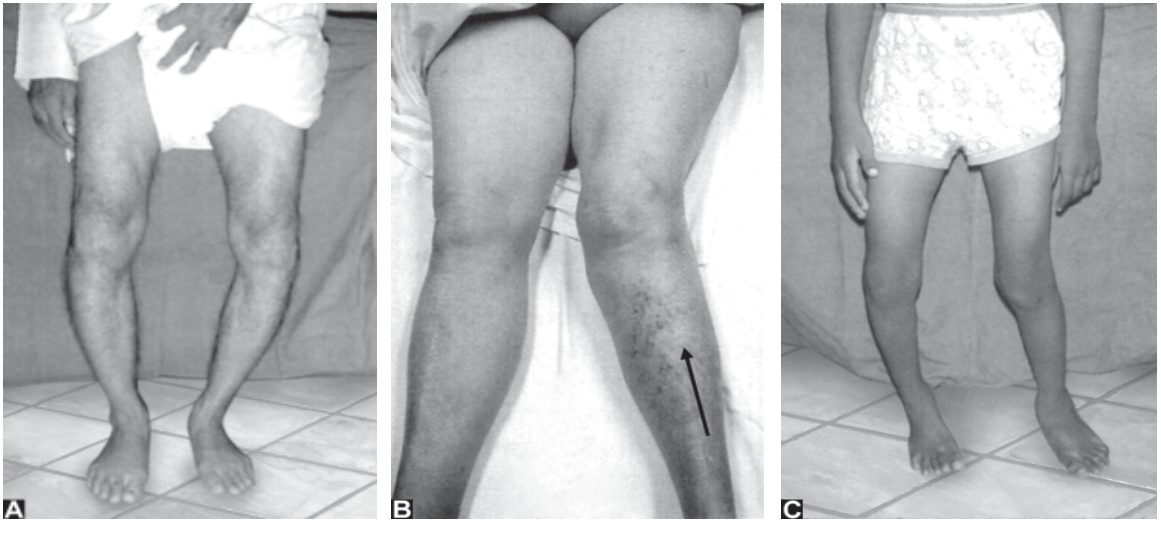
Inspect the popliteal area for any obvious swelling (Baker's cyst, gastrocnemius-semimembranosus bursa, popliteal artery aneurysm, lymphadenopathy, soft tissue tumours), wasting of hamstrings and calf muscles.

Supine on Couch

Confirm the inspection findings.

Feel

Feel for the quadriceps bulk and tone.



Figs 12.2A to C: Varus/valgus knee/windswept deformity

Warmth

This is felt using the dorsum of the hand and compared to the other side. Inflammatory conditions and infections produce increased warmth.

Tenderness

Ask the patient for tender spot and always look at the patient's face. Soft tissue and bony tenderness must be assessed individually.

In supine position with knee extended start from suprapatellar pouch, patella- retropatellar area (medial and lateral facet palpation by gliding the patella, to one side in extended knee and feeling the undersurface) and parapatellar area, patellar tendon, tibial tuberosity, condyles of femur and tibia especially the attachment of collateral ligaments.

Bend the knee to 90° and feel for tenderness along the medial and lateral joint line or thickening of bone due to osteophytes. Deep tenderness at medial femoral condyle just medial to patellar tendon in extreme flexion can be elicited in osteochondritis dissecans of medial femoral condyle. The localization of pain gives a clue to the probable pathology **(Figs 12.3 to 12.5)**.

Swelling

Localized or diffuse swelling should be examined in the way as described in Chapter 1. The generalized synovial hypertrophy is best felt over the anteromedial aspect of the suprapatellar pouch as it is immediately below the vastus medialis muscle and this muscle atrophies first in any knee pathology. The hypertrophied synovium can be rolled under the palpating fingers, has a doughy feeling and is tender. The fluid collection in the joint (effusion) can be assessed by the following manner.

Mild Effusion

Wipe or Bulge test: Small effusion is assessed by emptying the suprapatellar pouch by squeezing from the top. Now press medial to the patellar tendon and look for ripple of fluid appearing on the lateral aspect of the tendon.

Moderate Effusion

This can be assessed easily by cross fluctuation in two planes, above-downwards and mediolateral fluctuation.

Moderate to Severe Effusion

Patellar tap—Squeezing the suprapatellar pouch and giving a sharp tap on the patella with fingertips elicit patellar tap. The patella touches the trochlea with a knock and bounces back. This can be felt in moderate to severe effusion. It may be difficult to elicit in very tense effusion **(Fig. 12.6)**.

Tense effusion must be aspirated to relieve pain, to prevent quadriceps inhibition and for a diagnosis. Hemarthrosis can be due to anterior cruciate ligament injury, osteochondral fracture (usually in patellar subluxation or dislocation) or any intraarticular fracture, peripheral tear of meniscus, or capsuloligamentous complex injury, hemophilia, pigmented villonodular synovitis, etc. Presence of marrow fat (oil droplets) in hemarthrosis indicates intra-articular fracture.

Defects or Gaps

Feel for any defect in the quadriceps mechanism due to rupture. Similarly a patient with lateral joint problem should be palpated for intact fibular collateral ligament by keeping the leg bent to 90° and externally rotating the hip. A tight band could be felt spanning across the lateral femoral epicondyle to the head of fibula.

Distal Pulsation

Check for dorsalis pedis and posterior tibial artery pulsations.

Move

The normal movement is flexion and extension of the knee but a jog of adduction or abduction

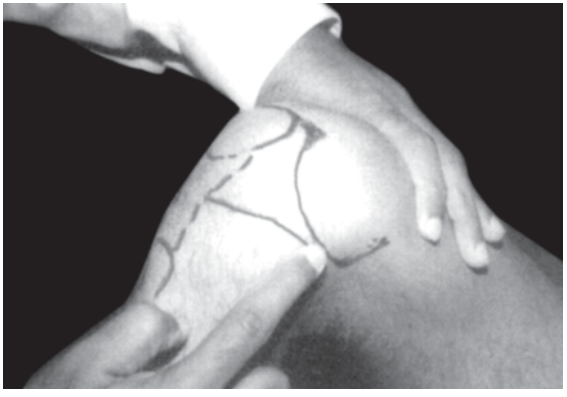


Fig. 12.3: Feeling the joint line

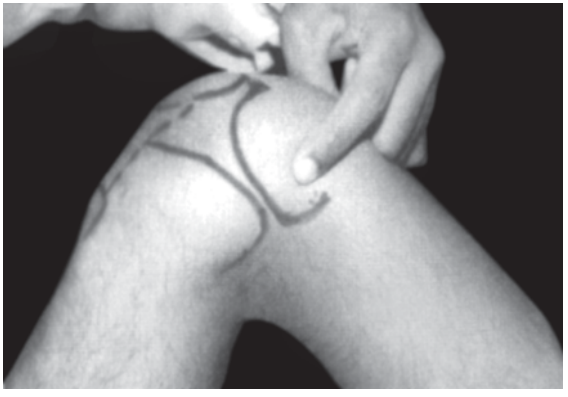


Fig. 12.5: Feeling for tenderness at femoral attachment of MCL

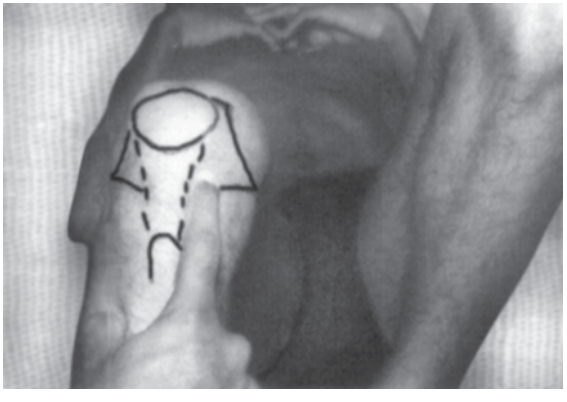


Fig. 12.4: Feeling for tenderness in fat-pad lesion

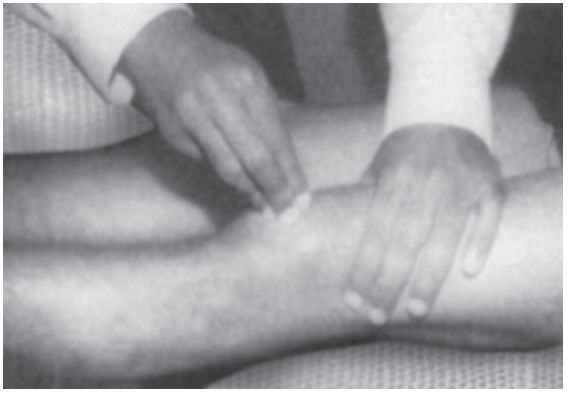


Fig. 12.6: Eliciting patellar tap

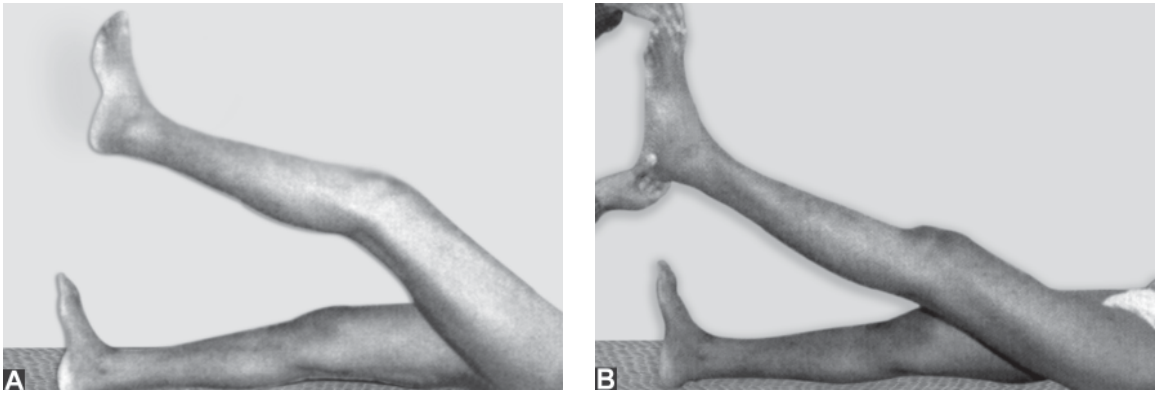
and some amount of internal and external rotation is possible (Normal range, Flexion—0 to 130° and Extension—0°).

Straight leg raise: Active extension is assessed by this test to verify the integrity of extensor mechanism. This may not be possible in quadriceps rupture, patella fracture and patellar tendon rupture. Lack of terminal extension can be due to fixed flexion deformity or extension lag. The quadriceps lag is assessed by the ability to passively extend fully **(Figs 12.7A and B)**. Active flexion is assessed by patient bending both knees fully and looking for heel to buttock distance **(Fig. 12.8)**.

Passive extension is assessed by lifting both heels to know fixed flexion deformity or recurvatum of the knee. The popliteal angle is the angle subtended by the long axis of thigh and lower leg segment, and gives the measure of fixed flexion deformity. Passive flexion with one hand on the knee is used to assess for crepitus or any loose body movement.

Measure

The girth of the thigh muscles from a fixed point above the knee is measured to know the amount of wasting. The thigh and leg segment can be measured to analyse leg length discrepancy.



Figs 12.7A and B: (A) Extension lag-knee; (B) Passive correction to full extension

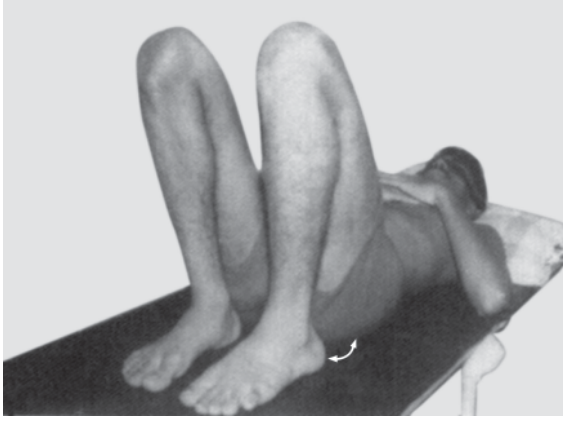


Fig. 12.8: Full knee flexion-heel to buttocks

SPECIAL TESTS TO ASSESS JOINT STABILITY

Examine the normal side first and compare with affected side.

Varus/Valgus Stress Test

In full extension assess for opening of the joint by holding the knee with one hand and the ankle with the other hand to apply varus (adduction)/valgus (abduction) stress. Abnormal opening of the joint indicates collateral ligament injury, posterior capsular tear and cruciate ligament tear. This also helps to know the correction of mediolateral deformity.

Varus or Valgus Stress Test in 30° Flexion (Figs 12.9 and 12.10)

Varus or valgus stress test in 30° flexion is used to assess isolated collateral ligament injury. This position relaxes the posterior capsule and cruciate ligaments. In heavy patients it is difficult to keep the knee flexed at 30° with one hand, so either we can place a pillow or bring the leg out by the side of the couch and bend the knee to 30° and perform the test. The minimal opening of the joint with pain on stressing indicates partial tear. Less pain and swelling with gross opening indicates complete tear of the ligament. Feel for the end point.

Grading of MCL injury with valgus stress testing:

Grade 1: 1 to 4 mm opening
Grade 2: 5 to 9 mm opening
Grade 3: 10 to 15 mm opening

Lachman's Test (Fig. 12.11)

With knee in 15 to 20 degrees flexion, examiner's one hand fixing the lower thigh and with the other hand thumb over the anterior joint line with all the fingers around the back of upper tibia perform a forward movement of the tibia on the femur and assess for the anterior glide and the end point. A mushy end point with forward movement of tibia on femur in excess of the

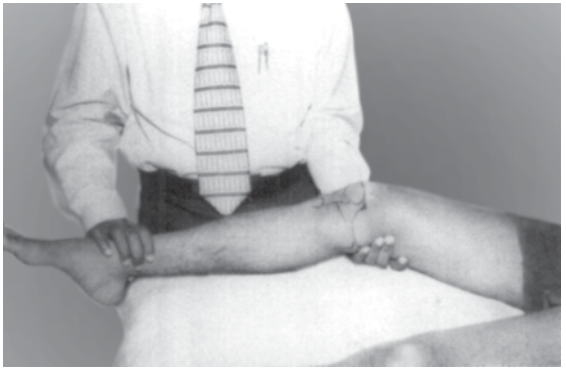


Fig. 12.9: Varus stress test

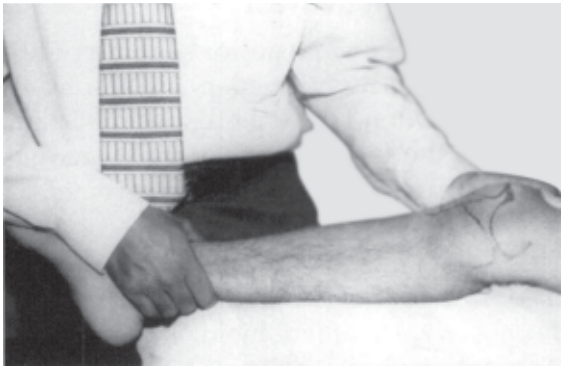


Fig. 12.10: Valgus stress test

normal side indicates anterior cruciate ligament rupture.

Grade 1+ (0-5 mm displacement)
Grade 2+ (5-10 mm displacement)
Grade 3+ (> 10 mm displacement)

Both the above tests can be easily performed in an acutely injured knee without bending and causing pain.

Posterior Sag

Bend both knees to 90 degrees and look tangentially from side for posterior sag of upper tibia. This indicates posterior cruciate ligament rupture. From this position bring the tibia to neutral to perform drawer tests **(Fig. 12.12)**.

Quadriceps Active Test

In the presence of posterior sagging of tibia in the above mentioned position, ask the patient to extend the knee against resistance by fixing the foot to the floor. This produces visible shift of the sagging tibia forwards.

Anterior Drawer Test (Fig. 12.13)

Sit on the foot of the patient with both the thumbs over the anterior joint line and fingers over the back of the upper tibia, feel for relaxed hamstrings and do forward movement of tibia on femur and assess the amount of forward displacement and the end point in comparison to the normal knee. This indicates ruptured anterior cruciate ligament.

The test is done in neutral position of the foot, in internal rotation and external rotation position of the foot.

Positive anterior drawer test in internally rotated foot indicates anterolateral instability (anterior cruciate ligament, lateral collateral ligament and arcuate ligament complex insufficiency).

Positive anterior drawer test in externally rotated foot indicates anteromedial instability (anterior cruciate ligament, medial collateral ligament, medial capsule and posterior oblique ligament insufficiency).

Posterior Drawer Test

This is performed in similar way but giving a posterior force on upper tibia to assess abnormal backward movement. Positive test indicates posterior cruciate ligament rupture.

Macintosh's Pivot Shift Test

Patient supine with one hand holding the upper tibia and other hand on the heel, with knee in full extension, internally rotate and apply valgus force. It will sublux the tibia in anterolateral instability. From extension maintaining the valgus—internal rotation force flex the knee. At about 30° flexion, the tibia reduces back with a clunk. This is due to

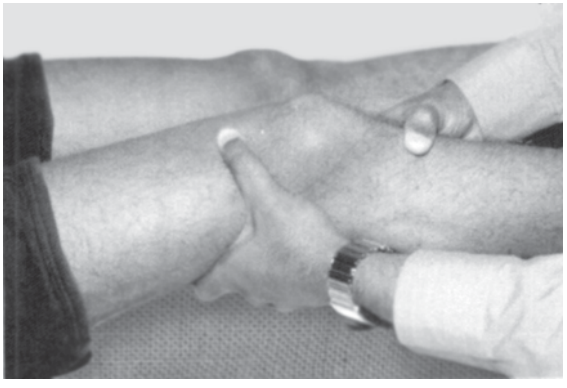


Fig. 12.11: Lachman's test

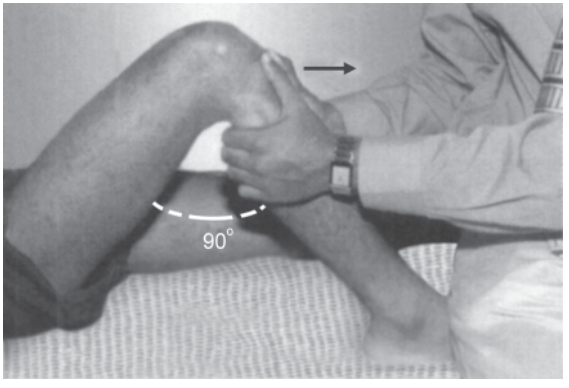


Fig. 12.13: Anterior drawer test

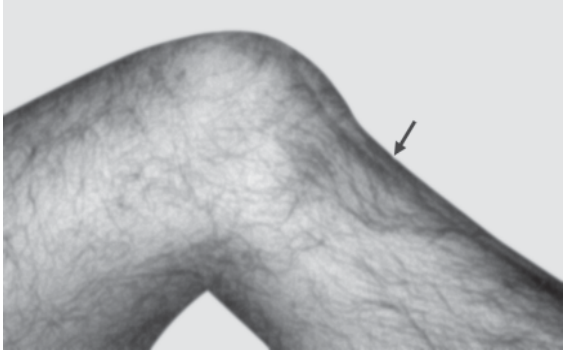


Fig. 12.12: Posterior sag in PCL insufficiency

the action of iliotibial band, which lies to the front of the knee axis in extension and on bending it falls behind the knee axis pulling the tibia to reduced position **(Figs 12.14A and B)**.

Jerk Test of Hughston and Losee

Patient supine with one hand holding the foot of the patient and the other hand over the proximal tibia starting from flexed position of the knee, extend the leg with valgus stress and internal rotation. The tibia subluxes with a jerk at 30° flexion.

External Rotation Recurvatum Test

Holding both legs with big toe and lifting the legs up can produce sagging of the knee with varus deformity and external rotation in posterolateral instability **(Fig. 12.15)**.

Reverse Pivot Shift Test of Jakob

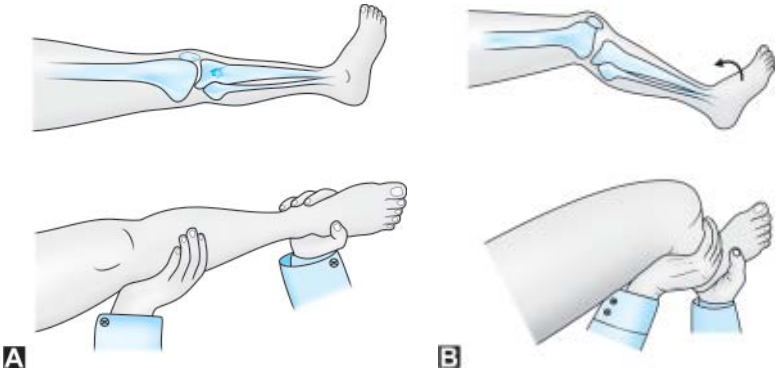
Patient supine, keep the knee flexed and leg externally rotated to sublux the tibia posterolaterally, and now extend the knee with slight axial load in valgus stress. The subluxation reduces with a jerk. This indicates posterolateral instability.

Dial Test

This test is for posterolateral instability. Passive external rotation of tibia with knee in 30° and 90° of flexion. Best performed with patient in prone position where posterior subluxation more at 30 degrees and less at 90° indicates isolated posterolateral corner injury. Marked subluxation with external rotation at both 30° and 90° indicates both PCL and posterolateral corner injury. Posterolateral instability is also confirmed by posterior drawer at 20° and varus stress test being positive.

McMurray's Test

This rotatory test was described to assess medial meniscus tear but also can be used for lateral meniscus. Flex the knee fully, externally rotate, apply valgus stress and extend the knee, any pain over the medial joint line or a click indicates probable medial meniscus tear.



Figs 12.14A and B: Pivot shift test

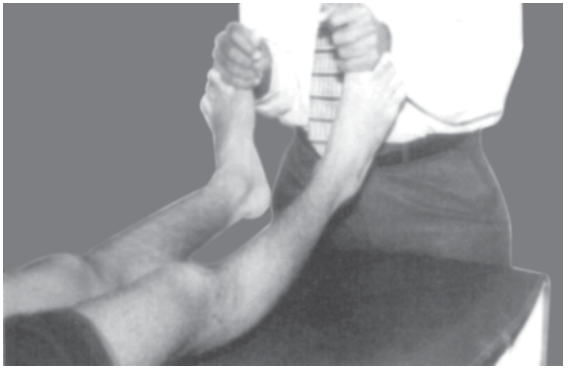


Fig. 12.15: External rotation-recurvatum test

By maneuvering this way we are producing a suction force to displace an unstable medial meniscal tear which gets caught between the articular surfaces and causes pain.

Similarly for testing lateral meniscus apply varus force, internally rotate the tibia and extend the knee from full flexion. The pain starting in full flexion indicates posterior horn pathology and pain in more extension indicates middle or anterior horn pathology.

Because of poor sensitivity a negative test does not rule out meniscal tear **(Fig. 12.16)**.

Prone on Couch

Look, feel and assess the popliteal area for tenderness, swelling and pulsation.

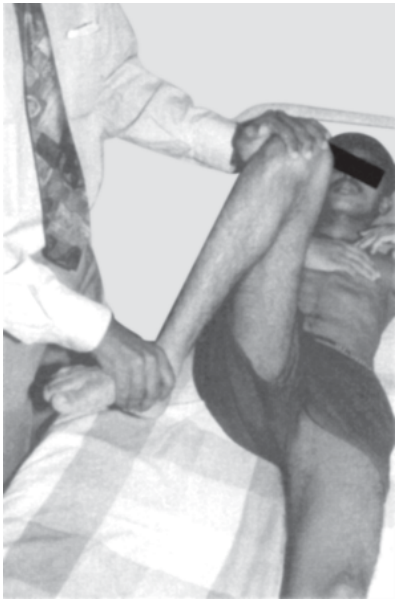


Fig. 12.16: McMurray's test

Apley's Grinding Test

Place the patient prone with knee joint flexed 90°, give axial compression and lateral rotation to the leg from the foot, if the patient complains of pain there may be tear of medial meniscus. With axial compression and internal rotation if pain appears, there may be lateral meniscus tear.

The same test if done in distraction of the joint may detect collateral ligament tear, lateral

rotation for medial ligament pathology and medial rotation for lateral ligament pathology.

PATELLOFEMORAL JOINT PROBLEMS

Usually a problem of young adults who give history of anterior knee pain, difficulty in getting up from prolonged sitting posture due to pain, difficulty in climbing stairs, inability to squat in the toilet, difficulty in kneeling or sitting cross legged on the floor.

Salient features to be noted are:

Patellar Rotation in Standing Position

Comment on squinting, tilt, foot rotation, wasting of quadriceps (VMO) and any lateral scars.

With Patient Lying

With patient lying assess patellar tracking on bending movement. Persistent lateral movement in knee flexion is called J sign (normally inferomedial).

Generalized Ligament Laxity Assessment (Figs 12.17A to C)

- Thumb touches the volar aspect of forearm.
- Little finger hyperextends parallel to forearm.
- Elbow hyperextends more than 15°.
- Knee hyperextends more than 15°.
- Palm to touch the floor by bending forward with the knee extended
- Presence of 4 or more signs indicates generalized ligament laxity.

Q Angle (Quadriceps Angle)

Knee in 20° flexion, either legs crossed or over a pillow, an imaginary line is drawn from ASIS to center of patella and from there to tibial tuberosity. Angle formed by these two lines is the Q angle. Normal range is 8 to 10° in males and 12 to 15° in females. Abnormal if more than 15° in males and more than 20° in females **(Fig. 12.18)**.

Now feel for the size of the patella, tenderness in the parapatellar and retropatellar surface, evidence of patella alta (high riding patella) or baja (low riding patella) by measuring the height of the patella and the length of patellar tendon in 30° flexion of the knee. Normally these are of same length.

Osmond-Clarke's Test (Fig. 12.19)

With gentle pressure in superior pole, ask the patient to lift the leg up straight without bending the knee. This produces contraction of quadriceps and shear at patellofemoral joint. This is a painful test similar to patellar grind where the patella is compressed on to trochlea.

Patellar Tilt Test

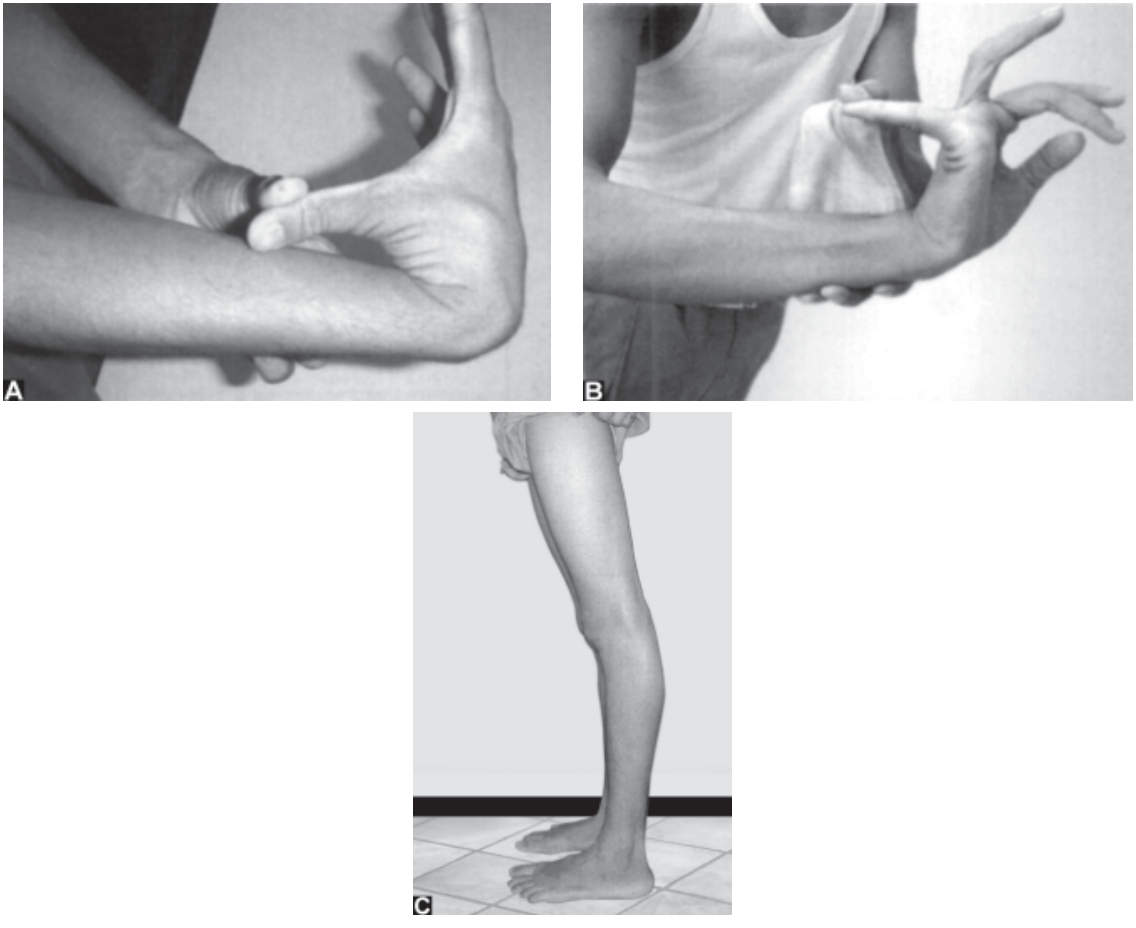
With the patient supine with knees extended compare transepicondylar axis to patellar tilt. Now elevate the lateral edge of patella and depress the medial edge. Normal is 0 to 20° tilt. Abnormal is not able to tilt beyond 0°(horizontal) because of lateral retinacular tightness. This may be helped with lateral release **(Fig. 12.20)**.

Patellar Glide Test (Sage Test)

With the knee flexed to 30° by crossing over the leg, the ability to translate patella medially and laterally is assessed. By dividing the patellar width into 4 quadrants, it is graded in number of quarter widths the patella glides. Normal lateral glide is upto 2.5 quadrants, more than 3 quadrants indicates abnormal medial restraint. Normal medial glide is 1 to 2.5 quadrants, less than 1 quadrant glide indicates tight lateral restraint. More than 3 quadrants is hypermobile patella.

Apprehension Test

This is done in 30° flexion of the knee with patient relaxed and patella pushed laterally. The test is positive if the patient is apprehensive and uncomfortable. This will be positive in recurrent dislocation or subluxation of patella **(Fig. 12.21)**.



Figs 12.17A to C: Ligament laxity tests

Assessment of Rotational Profile

As discussed in Chapter 14 assess for increased femoral anteversion and external tibial torsion.

SPECIAL NOTE

Genu Valgum (Knock Knees) (Fig. 12.22)

Trauma, infection (pyogenic, tuberculosis, postpolio), inflammation and metabolic or developmental disorders can cause this deformity. The intermalleolar distance is used to assess the progress of the deformity. The angle subtended by the anatomical longitudinal axis of thigh and the leg segment measured with goniometer gives the valgus angle. This can be accurately measured in standing radiograph.

To know whether the deformity is in femoral or tibial component assess the deformity in full flexion and full extension. Most of the times it is due to abnormal lateral femoral condyle. So the deformity is obvious in extension and disappears in flexion. This is due to different surface of femoral condyle articulates with the same surface of the tibia. In tibial defects the valgus deformity persists in all degrees.

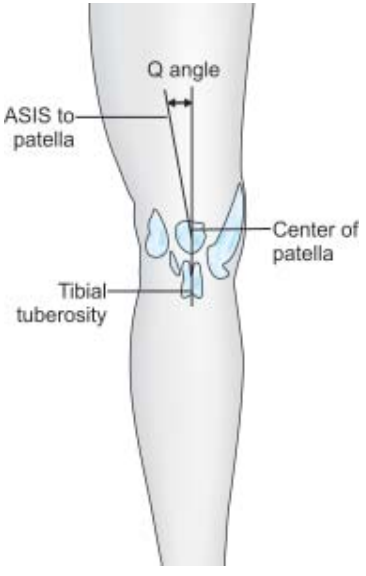


Fig. 12.18: Q angle

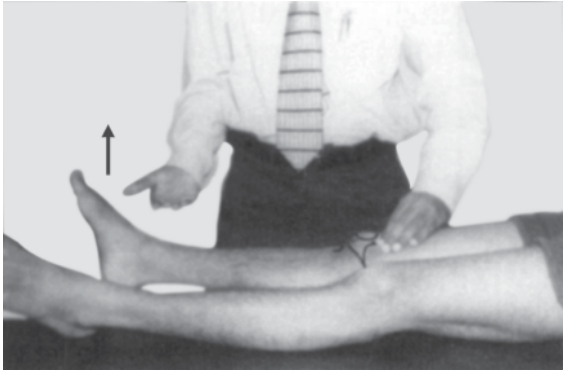


Fig. 12.19: Osmond-Clarke's test

This condition should also be assessed for common peroneal nerve palsy. The patient may have patellofemoral malalignment due to lateral position of tibial tuberosity and hyperpronated flat feet.

Genu Varum (Bow Legs)

Newborn baby is born with physiological varus, which tends to correct by 15 to 18 months and then approaches to more valgus. The valgus may

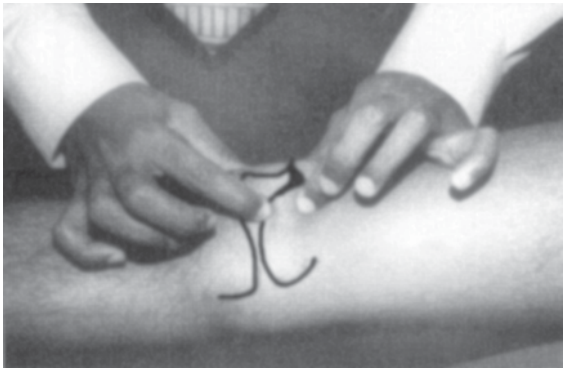


Fig 12.20: Patellar tilt

Fig. 12.21: Apprehension test

increase beyond the normal (6° to 8°) and then reduces to normal value at the age of 5 to 7 years. Unilateral deformity or severe varus deformity can be pathological. Always examine the patient standing to know the severity and standing X-ray is preferable. The distance between the knee joint or the angle between the thigh and leg segment can be used to assess the progress. Radiological measurement of varus angle is more accurate.

In kids with genu varum the differential diagnosis includes rickets, metaphyseal dysostosis, hypophosphatasia, Blount's disease and other dysplasias, trauma and infection. In elderly the most common cause is medial compartment osteoarthritis **(Figs 12.23A and B)** and rarely Paget's disease **(Fig. 12.24)**.

Genu Recurvatum

It is hyperextension of the knee or posterior bowing of the knee. This can result from damage to anterior portion of distal femoral or proximal tibial epiphysis due to trauma, infection or dysplasia; muscle weakness as in polio, cerebral palsy or arthrogryposis; congenital joint laxity and Charcot's disease. In young girls the growth of the proximal tibial epiphysis may be retarded from ballet dancing. This condition can cause patellofemoral malalignment. Look from the side with the patient standing to assess the degree of hyperextension at the knee. In fixed equinus of the ankle there may be associated recurvatum at the knee **(Figs 12.25A and B)**.

Swellings around the Knee

Prepatellar (housemaid's knee) and infrapatellar (Clergyman's knee) bursitis—cystic swelling with inflammation that appears in front of patella or below the patella respectively.

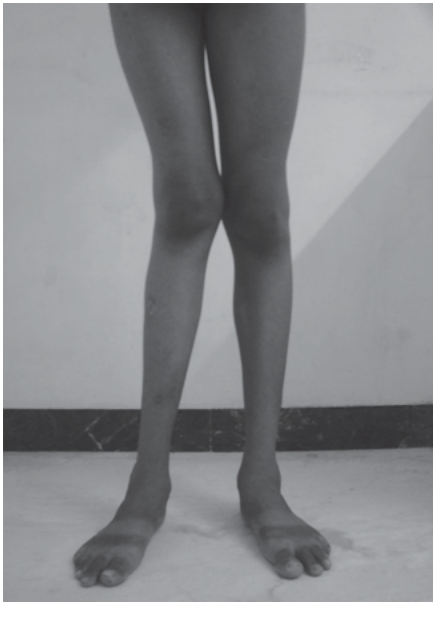
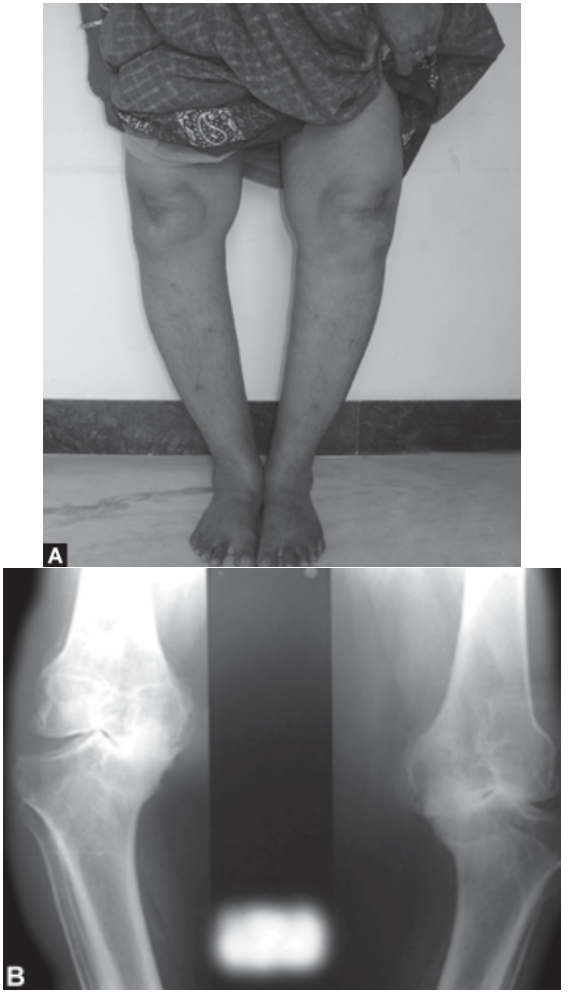


Fig. 12.22: Genu valgum: right leg *(For color version, see Plate 6)*

Medial and lateral swellings can be due to pes anserinus bursa, meniscal cysts, ganglion from superior tibio-fibular joint, and soft tissue or bony tumors.

Swelling in the back of the knee can be gastrocnemius-semimembranosus bursa—Baker's cyst. Baker's cyst is an outpouching due to collection of fluid from knee joint pathology (osteoarthritis or rheumatoid arthritis). It has to



Figs 12.23A and B: Severe osteoarthritis of knee resulting in bow leg *(For color version, Fig. 12.23A, see Plate 6)*

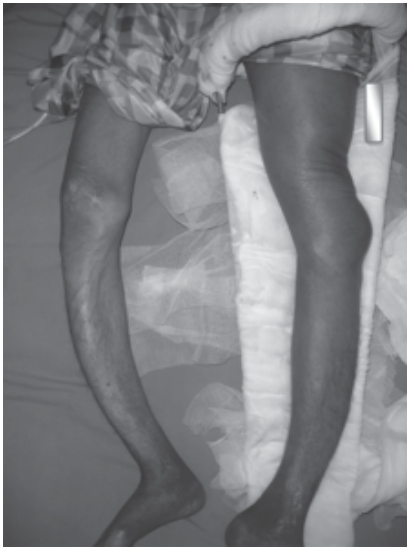
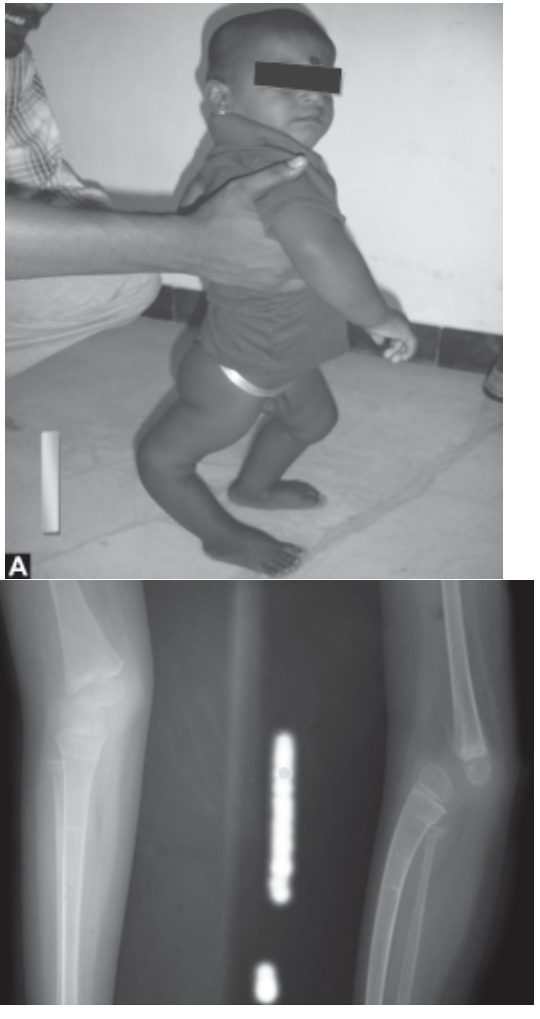


Fig. 12.24: Genu varum due to tibia varum from Paget's disease of right leg and fracture shaft of femur left leg *(For color version, see Plate 6)*

be differentiated from popliteal artery aneurysm—pulsatile nature and absence of transillumination. This can burst and the fluid can extravasate into calf to cause severe pain. Differential diagnosis for sudden onset severe pain in calf includes deep vein thrombosis, calf muscle rupture and stress fracture.

Osteoarthritis Knee

It is a degenerative wear and tear process of the knee joint. Primary osteoarthritis is common in elderly population and secondary osteoarthritis (trauma, infection, etc) can occur at any age. In the early stages it is usually the medial compartment (anteromedial) and later it becomes tricompartmental (lateral compartment and patellofemoral joint). Patient present with pain, swelling, deformity (varus deformity, flexion deformity), morning stiffness, limitation of movements and decrease in walking distance. A patient with fixed flexion deformity of knee often



Figs 12.25A and B: Genu recurvatum due to congenital anterior knee dislocation *(For color version, Fig. 12.25A, see Plate 7)*

complains of thigh pain due to constant action of quadriceps on standing resulting in muscle fatigue. Clinically joint line tenderness with bony thickening from osteophytes and limitation of movements can be appreciated. Weightbearing X-ray will show narrowing of joint space, subchondral sclerosis, marginal osteophyte formation, cyst formation and deformity.

Inflammatory Arthritis

This can affect knee joint producing swelling, pain, and limitation of movements. It can be rheumatoid or non-rheumatoid arthritis. Inflammatory arthritis of knee produces synovial hypertrophy and thickening with loss of joint space in X-ray and periarticular osteoporosis. It normally produces valgus deformity of the knee. Rheumatoid is diagnosed by polyarticular symmetrical involvement of small joints of fingers and by the criteria designed by American Rheumatoid Association. Non-rheumatoid arthritis can be of reactive arthritis (gonococcal or non-gonococcal urethritis), psoriatic arthropathy or other seronegative arthropathy.

Crystal Arthropathy

Calcium hydroxyapatite crystal arthritis or calcium pyrophosphate arthritis (Pseudogout) or Ochronosis (Alkaptunuria) are some of the conditions that cause sudden onset pain and inflammation. It is most common in elderly population with acute flare of pain. X-ray may show calcification of meniscus and articular cartilage. Aspirate can demonstrate the type of crystal causing inflammation of the joint.

Osteonecrosis

This condition again affects mostly elderly population with sudden onset severe pain in the knee (similar to myocardial infarction), with localized tenderness over the condyles and effusion of the joint. Patient may be unable to weight bear during this crisis period with rest pain. Cause is unknown. This must be differentiated from stress fracture of tibia (upper tibia) from severe varus deformity in osteoarthritis knee which is again sudden onset pain with inability weight bear and walk. Here pain is characteristically present on movement like any fracture.

Patellofemoral Conditions (Anterior Knee Pain) (Table 12.1)

Patellofemoral pathology causes pain especially on bending activities (bending more than 30° increases the load on patellofemoral joint and any lesion or malalignment can cause discomfort or pain). These patients find difficult to squat or get up from squatting position or to kneel or to sit cross-legged. Sitting on a chair for long time and getting up can cause anterior knee pain ' Cinema sign'. Difficulty in getting up and coming down stairs is again a problem. Clicking sensation with or without pain is common in patellofemoral malalignment. Reactionary effusion may be present in acute situation. Feeling of giving way due to subluxing or dislocating patella can be one of the presentation. Tenderness may be elicited at parapatellar area, retropatellar—medial and lateral facets, and over the patellar tendon (patellar tendinitis in runners). Other signs must be assessed in case of malalignment as discussed before.

Meniscal Injury

This occurs in two groups; young adult and middle age group. Mechanism of injury is due to twisting of the knee in flexed position. This happens often in sports person. In middle age adults degenerative tear of meniscus is common with trivial injury of the knee. Patient present with sharp localized pain over the joint line especially on twisting or turning movements, locking in bucket handle tear, inability to extend fully in displaced bucket handle tear and sometimes getting relieved of pain on slight manoeuvering of the joint.

Localized joint line tenderness along the meniscus, reactionary effusion, and positive McMurray's test are pathognomonic of meniscal injury. A negative McMurray test does not rule out meniscal tear.

Cruciate Ligament Injury

Anterior Cruciate Ligament Injury

A noncontact pivoting injury in sports or domestic accidents are commonly associated with a "pop" sound or feeling, with immediate swelling (haemarthrosis). It happens in flexion rotation mechanism of knee or in hyperextension or in frank dislocation. In chronic insufficiency patient describes

Table 12.1: Classification of patellofemoral disorders (Anterior knee pain)

I. Trauma
- A. Acute trauma
  1. Contusion
  2. Fracture
     - a. Patella
     - b. Femoral trochlea
     - c. Proximal tibial epiphysis (tubercle)
  3. Dislocation
  4. Rupture
     - a. Quadriceps tendon
     - b. Patellar tendon
- B. Repetitive trauma (Overuse syndromes)
  1. Patellar tendonitis (jumper's knee)
  2. Quadriceps tendonitis
  3. Peripatellar tendonitis (e.g. anterior knee pain of the adolescent due to hamstring contracture)
  4. Prepatellar bursitis (housemaid's knee)
  5. Apophysitis
     - a. Osgood-Schlatter disease
     - b. Sinding-Larsen-Johansson disease
- C. Late effects of trauma
  1. Post-traumatic chondromalacia patellae
  2. Post-traumatic patellofemoral arthritis
  3. Anterior fat pad syndrome (post-traumatic fibrosis)
  4. Traumatic neuralgia of cutaneous nerves
  5. Reflex sympathetic dystrophy of the patella
  6. Patellar osseous dystrophy
  7. Acquired patella infera
  8. Acquired quadriceps fibrosis

II. Patellofemoral dysplasia—patellofemoral malalignment
- A. Lateral patellar compression syndrome (LPCS)
  1. Secondary chondromalacia patellae
  2. Secondary patellofemoral arthritis
- B. Chronic subluxation of the patella (CSP)
  1. Secondary chondromalacia patellae
  2. Secondary patellofemoral arthritis
- C. Recurrent dislocation of the patella (RDP)
  1. Associated fracture
     - a. Osteochondral (intra-articular)
     - b. Avulsion (extra-articular)
  2. Secondary chondromalacia patellae
  3. Secondary patellofemoral arthritis
- D. Chronic dislocation of the patella
  1. Congenital
  2. Acquired

III. Idiopathic chondromalacia patellae

IV. Osteochondritis dissecans
- a. Patella
- b. Femoral trochlea

V. Synovial plicae (anatomic variant made symptomatic by acute or repetitive trauma)
- a. Medial patellar (shelf)
- b. Suprapatellar
- c. Lateral patellar

a giving way feeling or abnormal movement of thigh bone over leg bone and an insecure feeling.

Lachman's test is most sensitive and easily done in acute swollen knee as there is no need to bend the knee more than 20°. In chronic insufficiency Lachman's test, anterior drawer test and pivot shift test may be positive. Associated meniscal injury is common.

Posterior Cruciate Ligament Injury

This most commonly results from a direct bow to the upper tibia in a flexed knee-dashboard injury or hyperflexion without a blow or in frank dislocation. Loss of confidence in the knee and giving way feeling may be present. Positive posterior sagging, posterior drawer test, external rotation recurvatum test and posterolateral instability tests may be present.

Osteochondritis Dissecans

Osteochondral lesion of bone and overlying cartilage resulting in separation and loss of blood supply usually involves the lateral aspect of medial femoral condyle and is common in teenagers and young adults. It can affect lateral femoral condyle and patella. The lesion is thought to be due to occult trauma, ischemia or abnormal epiphyseal ossification. Patients present with pain, swelling or mechanical symptoms. Localized tenderness in fully flexed knee over the femoral condyle area may be present. Tunnel or notch view X-ray is valuable for identification. Children have the best prognosis.

Osgood-Schlatter Disease

Osteochondritis of tibial tubercle apophysis due to stress from extensor mechanism in a growing child. There is localized pain and tenderness over tibial tuberosity with prominent tubercle. X-ray may show fragmentation of apophysis.

Failed Total Knee Replacement

There is increasing number of patients who had total knee replacement coming back with pain, instability, swelling, infection and stiffness. A systematic approach to the evaluation of the patient requiring revision total knee arthroplasty can help identify the correct diagnosis and guide surgical intervention.

The causes of dysfunction and pain are considered in two broad categories: extrinsic (extra-articular) and intrinsic (intra-articular). Extrinsic sources of pain include the ipsilateral hip, lumbar spine (stenosis or radiculopathy), soft tissue inflammation (pes anserinus bursitis or iliotibial, patellar or quadriceps tendinitis), complex regional pain syndrome, neuroma, vascular claudication, stress fracture and rarely intrapelvic lesion compressing femoral cutaneous nerve. Intrinsic sources include aseptic loosening, polyethylene wear, osteolysis, malalignment, instability (mediolateral, flexion or global), infection, implant fracture, arthrofibrosis, soft tissue impingement, component overhang, and dysfunction of extensor mechanism like instability, fracture, maltracking, lateral patellar facet impingement, excessive component construct thickness, patella baja, and patellar or quadriceps tendon rupture.

Pain that was present before surgery persisted without change indicates extrinsic etiology. Pain that began within the first year after surgery suggests infection, malrotation, or soft tissue impingement. Pain after a year suggests wear, osteolysis, loosening or infection (acute hematogenous or late chronic). Comorbid conditions should be noted. Visual inspection and careful palpation for swelling and point tenderness are noted. Stability testing in extension, mid flexion and 90° flexion and evaluation of patellofemoral stability were done. Note the gait and alignment on walking, measure active and passive ranges of motion, evaluate patellar tracking, patellar clunk, neurovascular examination including the power of quadriceps and examine adjacent joints and opposite limb for completion.

13 CHAPTER

Examination of Ankle and Foot

The bipedal stance of human being makes our foot a unique structure to take the weight of the whole body. Any slight change in the biomechanics alters the weightbearing pattern and results in pain and deformity.

This chapter offers an introduction to the clinical assessment of the foot and ankle. The first section contains general instruction. There are then separate sections on the ankle and regions of the foot.

PRESENTING COMPLAINT

Define exactly what the patient is complaining of, how the problem first started, how long has it been going on and about the footwear.

Pain

Ask about duration, site, aggravating and relieving factors. Pain during the night, early morning or on walking after prolonged rest or sitting as in plantar fascitis, walking on uneven surface and climbing up or down stairs should be recorded.

Morning Stiffness

In inflammatory conditions like rheumatoid or seronegative arthritis.

Deformity

Onset, progress, problem with wearing footwear, cosmetic, associated callosities and pressure sores.

Swelling

Site, duration, onset—sudden or insidious, traumatic or postsurgical, localized or generalized, associated with inflammation or other swellings. Enquire about medical conditions, which may cause bilateral foot swelling: Renal or cardiac problems, anemia, hypoproteinemia, pregnancy, liver disease, lymphoedema, etc. In unilateral foot swelling other than local pathology think of any pelvic pathology causing venous stasis like gynecological problems and gather more information.

If there is an injury, ask about the mechanism of injury. This may indicate likely injuries. Fall from height and landing on heel may result in calcaneal fracture; twisting injury to feet can cause fracture base of 5th metatarsal and twisting injury to ankle can cause ligament sprain or fracture.

Giving Way

Can be due to ankle instability, anterolateral impingement syndrome, neurological problems or osteochondritis of dome of talus.

Neurological Symptoms

Weakness, numbness, and pins and needles.

Other Joint Involvement

Rheumatoid arthritis, seronegative-arthropathy and gout. It is a good habit to look at hands for joint involvement.

Miscellaneous Symptoms

Ulcer, gangrene—dry or wet, painful corns or callosities, web space skin problems—Athelete's

foot, ingrowing toenail with nail fold infection and pain.

Limitation of Activities

Effects on gait and mobility. How is the problem affecting the patient's life: Work, sport and hobbies, social activities.

PAST HISTORY

Does the patient have any other relevant medical or psychological conditions, e.g. diabetes, rheumatism, gout, tuberculosis, trauma or allergy? Is he or she on any regular medication?

FAMILY HISTORY

Do they have a family history of present problem or related conditions? If anything significant like rheumatism, idiopathic flat feet, generalized ligament laxity.

PERSONAL HISTORY

Occupation, hobbies, smoking, alcoholism.

TREATMENT HISTORY

What treatment has been tried and with what result. Ask about surgery, local injections, physiotherapy, splints and orthoses.

All patients with foot and ankle problems should be asked about:

1. Diabetes
2. Inflammatory arthropathy
3. Neurological disease
4. Vascular disease
5. Trauma.

What Sort of Treatment do they Expect and Want?

EXAMINATION

General Points

Avoid the habit of examining patients only from the ankle down. Many generalized diseases produce problems in the foot. The foot is a common presenting site of rheumatoid arthritis, for example. The general examination also allows assessment of a person's overall fitness. It will be tailored to the problems suggested by the history: for instance, examination of other joints in suspected arthritis, full neurological examination in a patient with suspected neuropathy, assessment of joint mobility in a child with flat feet, looking for features of a syndrome in a child with club feet.

Spine: Not only overt neurological disease but also features such as pes cavus, dysmorphic feet or toes, or marked foot asymmetry, should lead to a full examination of the spine and lower limb neurology. Look for scoliosis, evidence of spinal dysraphism such as sacral sinus, lipoma or hairy patch. Do a full neurological examination of the lower limbs, including evaluation of pressure, vibration and two-point perception-abnormalities in these may be the only features of diabetic neuropathy or tarsal tunnel syndrome.

Limb alignment and length: Look for pelvic obliquity, limb length discrepancy (and its level), valgus/varus deformities, usually at the knee, and rotational alignment. The differential amounts of internal and external rotation at the hip can be used to measure femoral rotation and the thigh-foot test with neutral hindfoot to measure tibial torsion. The overall foot position completes rotational alignment. Check for contractures of the hips and knees, especially in patients with neurological disease or arthritis.

Gait: Familiarize yourself with the gait cycle and get used to analyzing people's gait. Normal gait has stance phase (weight bearing) and swing phase (non-weight bearing). Stance phase consists of initial contact (normally heel strike), loading response, midstance, terminal stance and preswing (toe off). Swing phase consists of initial swing, midswing and terminal swing. At initial contact **(Fig. 13.1)** the body is about to begin deceleration. The ground reaction force is posterior to the ankle at or just in front of the

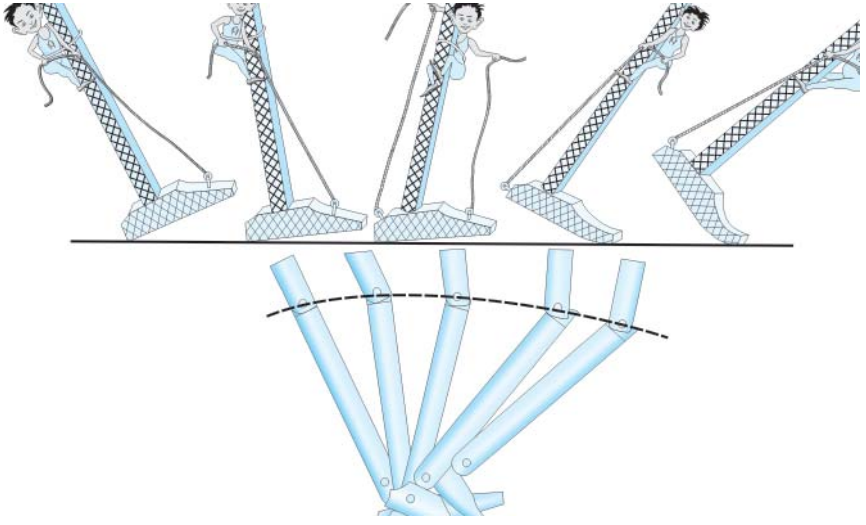


Fig. 13.1: Gait

knee and anterior to the hip joint. This produces a plantar flexion moment at the ankle, zero to slight extension moment at the knee and a flexion moment at the hip. These moments would be resisted by eccentric contraction of ankle dorsiflexors, posterior knee capsule and hip extensors. This is the first rocker at ankle.

The next illustration is at the end of loading response. Body weight has been decelerated by controlled knee flexion and ankle plantar flexion. The ground reaction force imposing a zero moment at the ankle as first rocker has been completed and second rocker is about to begin. Hence, anterior tibial muscle action ceases and triceps surae, tibialis posterior and peroneal action begins. There is a large flexion moment at the knee requiring quadriceps (vasti) contraction. Hip extensors are ceasing activity as the ground reaction force has moved anteriorly and is now passing through the hip.

In midstance, the ground reaction force passes anterior to the knee and posterior to the hip. Thus no muscle action is necessary at either joint since both can be stabilized by ligaments (iliofemoral ligament at the hip and posterior capsule and cruciates at the knee). Hence, by eccentric contraction of the soleus in second rocker the sagittal plane position of the ground reaction force is controlled, thus allowing one muscle to stabilize three joints. Initial swing has begun on the contralateral side. The body is in single support and its center of the mass has reached its highpoint.

Terminal stance: Begins as the body's mass moves in front of the base of support such that it is literally falling anteriorly and towards the unsupported side. Gastrocnemius (fast twitch) has joined soleus (slow twitch) with sufficient power to stop further dorsiflexion of the ankle. Hence the heel leaves the ground and the triceps surae are now contracting concentrically as third rocker begins. Acceleration and forward propulsion are produced by the combination of triceps action and forward fall of the trunk. By the end of terminal stance, the opposite limb is in terminal swing.

Initial contact of the opposite limb marks the beginning of double support and of preswing. Iliopsoas is now firing concentrically as an accelerator (flexor) of the thigh. As the ground reaction force moves behind the knee and weight

is unloaded onto the opposite limb, the ankle plantar flexes and the knee is driven into flexion. With normal cadence (number of steps per minute), no muscle action is necessary at the knee. However during fast cadence, the rectus femoris comes into action to provide an additional flexion force at the hip and to eccentrically decelerate knee flexion, i.e. prevent excessive heel rise. At normal cadence, ankle plantar flexion is approximately 27°, knee flexion 45° and hip flexion 10° at the time of toe-off. This sequence of events gets changed in abnormal gait.

Ask the patient to walk up and down while you concentrate on each phase of the cycle in turn—first contact, shock absorption, mid-stance and so on. You can do a lot of gait analysis with the naked eye and patience! Learn the common abnormal gaits and their clinical significance.

Shoes: All patients are asked to bring a well-worn pair of shoes or slippers to clinic. Examination of these is like a summary of gait over time. They show the areas under pressure in gait and from deformity, and the areas that take no pressure at all. They also show what forces have been exerted on the foot in the recent past. Sometimes changing a patient's ideas about shoe wear is the most important service we can offer them.

Skin: Look for inflammation, infection, varicose veins, tophi, discoloration, gangrene, scars or contracture, ulcers, calluses, corns, trophic changes, the cool dry hairless foot of vascular disease, the warm dry neuropathic foot.

Overall foot shape: Assess the size of both feet: Normal, small as in clubfoot or long and thin in Marfan's syndrome.

Examine the overall foot shape with the patient standing. The hindfoot component of foot shape is best appreciated from behind.

Recognize common foot shapes:

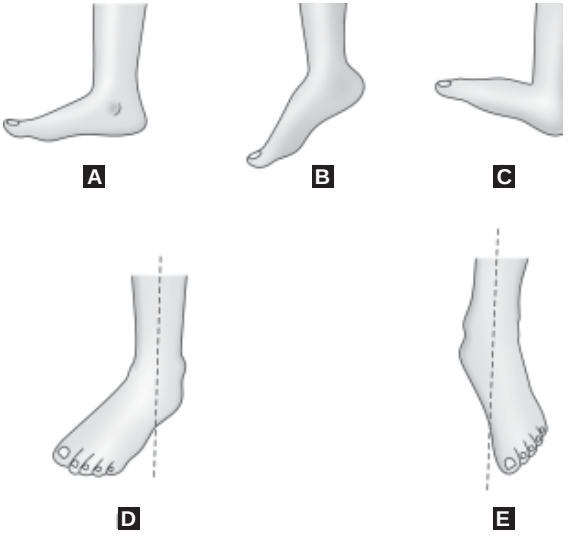
1. Neutral or rectus foot—No overall deformity.
2. Flat foot—Heel valgus, low arch, commonly forefoot abduction and supination. The subtalar joint is commonly in the overpronated position in stance and may be even more so on walking. Distinguish between flexible and rigid flat feet by asking the patient to stand on tiptoe to see if the arch re-appears and the heel goes into varus. Then do a single foot tiptoe test to look for tibialis posterior insufficiency. The "too many toes sign" demonstrates forefoot abduction. Manipulate the subtalar joint to identify a rigid hindfoot suggesting arthritis or a tarsal coalition. Exclude a neurological cause by appropriate examination.
3. Cavus foot—Typically with a plantar flexed first ray, high arch and forefoot pronation. In many cases the hindfoot is in varus and this may be fixed or mobile. Pes cavus may be associated with spinal anomalies (especially if asymmetrical) or with hereditary sensorimotor neuropathies such as Charcot-Marie-Tooth disease. Use the Coleman block test to tell the difference between fixed and mobile hindfoot varus. The cavus foot typically has a plantar flexed first metatarsal, producing a pronation deformity of the foot. To make the foot flat on the floor the hind foot inverts. In this special test a wooden block is kept under the heel and lateral rays so that the 1st ray is allowed to drop freely. This results in reproduction of forefoot pronation with the hind foot in neutral position. If the hind foot varus does not correct with this test, then it indicates fixed deformity of subtalar joint due to long-standing deformity.
4. Skewfoot—Hindfoot valgus and forefoot adduction. Do the same tests for hindfoot correction as in flatfoot. Manipulate the forefoot to determine correctability of adduction.
5. Metatarsus adductus—Neutral hindfoot and adduction of the metatarsus (some patients have some forefoot supination too). Commonly seen in pre-school children when it is usually correctable, but also in adults when it is often relatively fixed but usually in itself asymptomatic.

Neurological Examination

Neurological examination including strength testing, usually recorded in MRC grades, light touch, pinprick and pressure testing with Semmes-Weinstein filaments. Palpate and perform the Tinel test for neuromas over all major nerves, especially those which might explain the patient's symptoms (such as the tibial nerve in a patient complaining of pain and paraesthesia on the sole of the foot). The Romberg test—failure to keep the body balance upright on closing the eyes-indicates posterior column lesion and favours the diagnosis of Friedreich's ataxia.

Vascular Examination

Vascular examination includes, if necessary, ankle pressure measurements with the Doppler probe, and calculation of the ankle-brachial systolic index.



Figs 13.2A to E: Foot deformities: (A) Normal foot; (B) Equinus; (C) Calcaneus; (D) Varus, and (E) Valgus deformity

LOCAL EXAMINATION

Explain to the patient at each step what you are doing and follow a systematic approach.

Look

It is always better to start the examination with the footwear, which can give a clue to the problem.

Footwear

Look for irregular wear of the sole, the insole and appropriate fit.

Standing

Expose up to both knees to assess deformities. Start from front; look at sides and then the back of the feet.

From Front

Posture: Is the foot plantigrade or equinus or calcaneus? In-toeing, inversion or eversion of foot must be noted **(Figs 13.2A to E)**.

Foot size: Normal, small as in clubfoot or long and thin as in Marfan's syndrome.

Skin condition: Inflammation, infection, varicose veins, discoloration, gangrene, scars or contracture, ulcers, trophic changes.

Nail condition: In-growing toenail, nail fold infection—paronychia or fungal, pitting and scaling in psoriasis, trophic changes.

Swelling: Ganglion, exostosis, fractures/dislocation

Hair: Changes and distribution

Deformities

Toes-splaying, over-riding, clawing (hyperextension at MTPJ), mallet toes (flexion at DIPJ) or hammer toes (flexion at PIPJ)

Big toe-hallux valgus (outward deviation), hallux flexus (bent down), hallux varus (medial deviation) or bunion (prominence over Ist MTPJ)

Forefoot—adduction, abduction

Midfoot—supination, pronation

Muscle wasting Extensor digitorum brevis, a small bulky muscle seen in front of the anterolateral aspect of the ankle.

From Side

- Assess the arch of the foot for flat foot or cavus.
- Tibialis posterior tendonitis—swelling and inflammation from the navicular tuberosity along the posterior aspect of medial malleolus and above.
- Look for peroneal tendon standing out in spasm.
- Venous ulcer or pigmentation.

From Back

- Look for calf muscle wasting.
- Look for attitude of the heel—varus or valgus.
- Too many toes sign-more than two toes visible laterally due to planovalgus feet.
- Widening of heel in calcaneal fractures.
- Swelling posterior aspect of heel—insertional or noninsertional Achilles tendonitis, retrocalcaneal bursitis, rheumatoid nodules, lipoma, etc.
- Gap in heel cord in complete tendo-Achilles tear.

Standing on Tip Toes

Implies good strength of tendo—Achilles and also inverts the heel due to the action of tibialis posterior (invertor). Tibialis posterior insufficiency results in failure of inversion or inability to do single leg stance on tiptoes **(Fig. 13.3)**.

Standing on Heel

To assess the power of dorsiflexors.
Leg length discrepancy can be commented.

Walking

Assess for gait patterns.
1. Stiff ankle—peg-like gait
2. Foot drop—forefoot drops in swing and patient has high stepping to clear the ground and doesn't have heel strike on stance phase
3. Fixed equinus

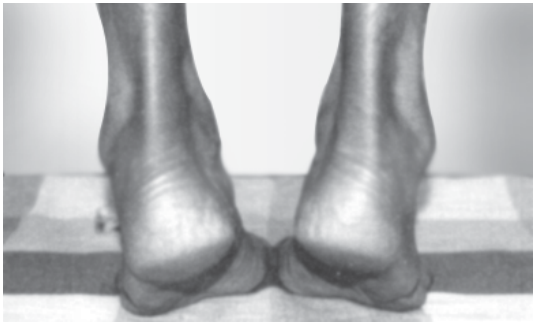


Fig. 13.3: Standing on tip toes shows heel inversion

4. Antalgic—patients with ankle or subtalar joint pain walk with foot externally rotated with short stance phase.

Lying Down

Look at the sole for callosities, corns, ulcers, and web-space infections—fungal infection.

Feel

Temperature, pulse, skeletal structure, joints, ligament and tendon course and insertions
Ask the patient for any tender spot and examine that part at the end. Always look at the patient when you palpate. Feel for warmth and then bony or soft tissue tenderness starting from ankle **(Fig. 13.4)** to toes including metatarsal heads (metatarsalgia) and intermetatarsal space (Morton's neuroma).

Swelling is examined as described in Chapter 1. In ankle effusion there may be fullness along the anterior joint line and on either sides of tendo-Achilles with cross fluctuation.

Deformity is assessed for flexibility, rigidity or for partial correctability. Heel is held square (in neutral) to assess the forefoot deformity (supination-pronation).

Gentle percussion over the peroneal tendon can elicit spasm of peroneal muscles.

Move

Starting from ankle, move onto subtalar joint (talocalcaneal), midtarsal joints (talonavicular

and calcaneocuboid), metatarsophalangeal joints and interphalangeal joint movements. Start with active movements in both feet and then test for passive movements.

Active and passive range of movements, stability, flexibility, and contractures of joints are identified.

Ankle dorsiflexion (normal range: 0 to 20°) can be assessed by asking patient to actively pull up the feet. Ask the patient to move foot down to assess plantarflexion (normal range: 0 to 60°) and in same way the inversion (normal range: 0 to 20°) and eversion (normal range: 0 to 10°) of subtalar joint is tested by actively moving the foot inwards and outwards respectively (or by asking the patient to touch the examiner's fingertip, held inside and outside, with their forefoot without moving at hips or knees).

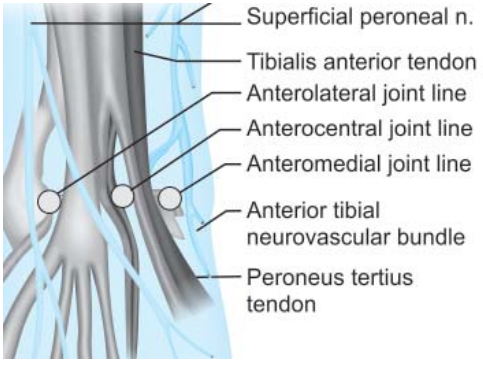


Fig. 13.4: Surface marking on anterior aspect of ankle

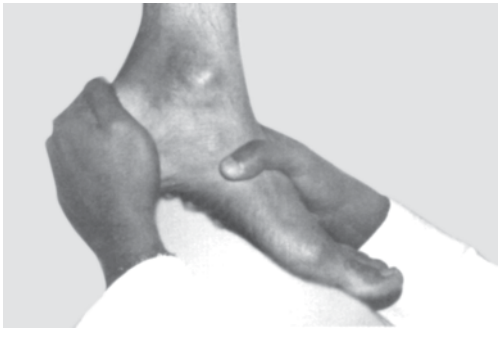


Fig. 13.5: Testing plantar flexion of foot

Active toe movements are next looked into.

Passive movements at ankle are tested by holding the heel in one hand and neck of talus with the other hand to check plantar flexion and dorsiflexion **(Figs 13.5 and 13.6)**. Limitation of dorsiflexion movement should always be checked with knee joint in flexion to rule out gastrocnemius tightness.

Subtalar movement is assessed by holding the heel by cupping with the hand, foot supported in neutral position with the examiner's forearm, test inversion and eversion **(Figs 13.7 and 13.8)**.

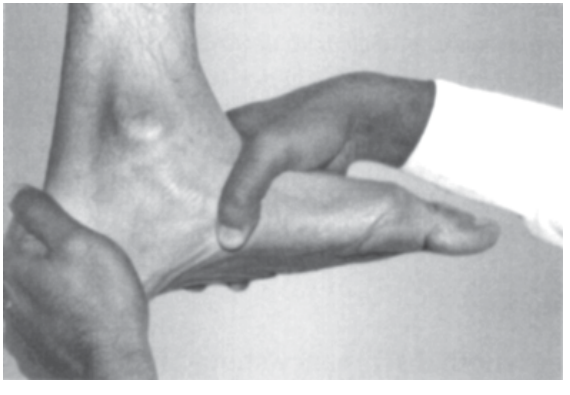


Fig. 13.6: Testing dorsiflexion of foot

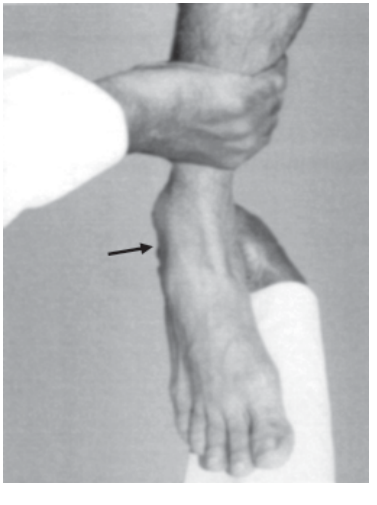


Fig. 13.7: Testing inversion of foot

Subtalar movement can also be tested by holding the neck of talus with the thumb and four fingers of one hand while the other hand holds the heel, keeping the foot in neutral position and doing inversion and eversion movements **(Fig. 13.9)**.

[General belief of dorsiflexing the foot to lock the talus to test subtalar movements is not needed as this restricts the normal subtalar movements because of tight medial tendinous (posterior compartment muscles) and lateral tendinous (peroneal compartment muscles) structures embracing the calcaneum. Moreover there is no inversion or eversion movement at the ankle hence there is no necessity to lock the talus].

Mid tarsal movements are tested by holding the talus with one hand and the other hand holding the forefoot to do supination and pronation.

Toe movements are individually tested from MTP to IP joints for dorsiflexion and plantar flexion.

Measure

Leg length discrepancy should be assessed.

Size of the foot:

On the medial side:

a. Tip of medial malleolus to heel tip.
b. Medial malleolus to 1st metatarsal head.

On the lateral side:

a. Tip of lateral malleolus to heel tip.
b. Lateral malleolus to 5th metatarsal head.

Neurological Assessment

i. Motor system: Assess ankle dorsiflexors, plantarflexors, subtalar joint invertors and evertors by MRC grading. Always feel the muscle to confirm the contraction. Assess the strength of the toe flexors and extensors.
ii. Sensory system: Examination of sensation is very important in neuropathic foot-diabetic foot, neurotrophic ulcers, spina bifida **(Fig. 13.10)**.
iii. Reflexes.

Vascular Assessment

Feel for dorsalis pedis and posterior tibial pulses, if feeble or absent feel the popliteal and femoral pulses. Absent pulse warrants further investigations before any foot surgery to prevent wound problems.

Look for changes due to ischaemia or gangrene.

Check for varicose veins in the leg, venous ulcer or deep vein thrombosis.

Examine knee For any deformities, which may cause secondary deformity in the foot.

Examine spine Neurocutaneous markers for spina bifida, deformity, etc.

Salient features in history and examination of each region of the foot and ankle are elaborated in detail as follows.

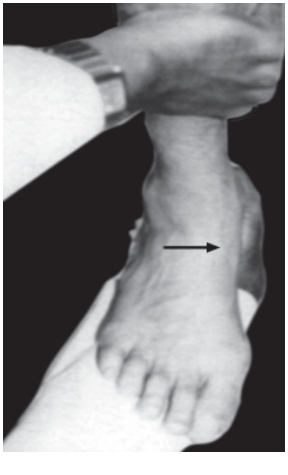


Fig. 13.8: Testing eversion of foot

Fig. 13.9: Alternative method of testing inversion/eversion

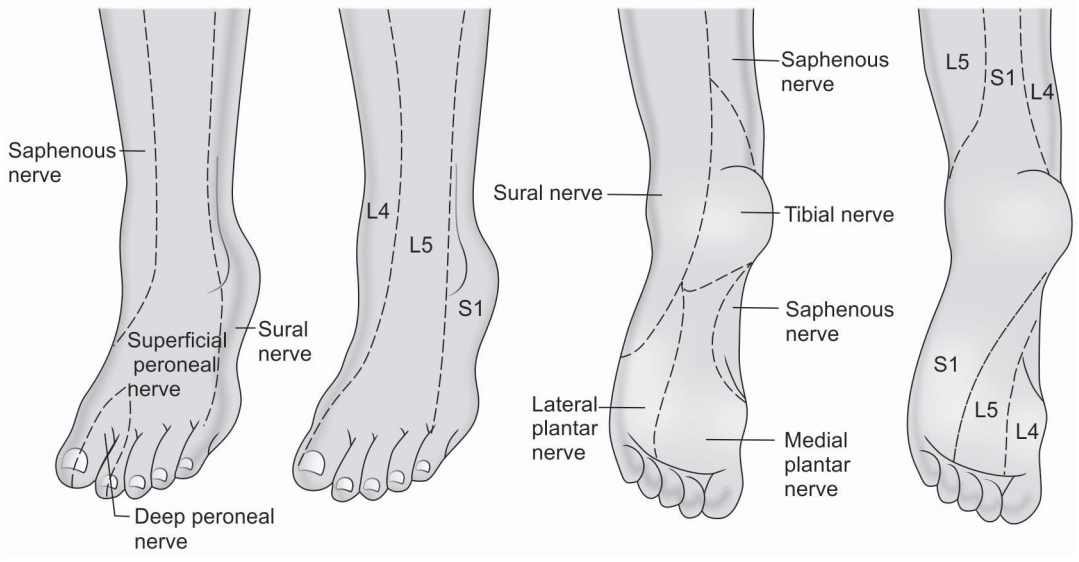


Fig. 13.10: Sensation in the foot

ANKLE

History: As well as asking patients with ankle problems about pain, stiffness, swelling and all the usual things, ask:

a. Does the ankle give way-how often, in what circumstances?
b. Does the ankle lock?
c. Does it feel as if something jumps or comes out of place in the ankle?

Inspection: Look at the ankle for swelling, redness, deformity, sinuses, scars.

Palpation: Feel the temperature of the joint and compare it with the other side. Feel for tender areas, systematically checking:

a. Anterior joint line **(see Fig. 13.4)**
b. Lateral gutter and lateral ligaments
c. Syndesmosis
d. Posterior joint line
e. Medial ligament complex
f. Medial gutter

Feel for an effusion, synovitis, deformity, bony prominence and loose bodies.

Range of movement: First get the patient to move the ankle through their active range of movement and compare with the other side, then repeat passively. Check particularly for loss of dorsiflexion as this is more disabling and may be related to other problems in the foot. Loss of ankle dorsiflexion is commonly seen after an injury but may also accompany Achilles tendon problems, arthritis or flatfoot. The actual block to dorsiflexion may be a tight Achilles tendon, anterior ankle impingement and incongruity or arthrofibrosis of the ankle. If the patient has anterior or posterior ankle pain, also check for impingement in the dorsiflexed (especially with the foot everted) or plantarflexed position. Local anesthetic injection may clarify this further.

Stability: Test for ankle stability using the anterior drawer and tilt tests. In the acute trauma situation pain makes these difficult. Sometimes local anaesthetic injection into damaged ligaments or the lateral popliteal nerve makes stress testing easier. The anterior drawer test should be done with the ankle plantarflexed 20°. Push posteriorly

with one hand on the tibial shaft and draw the calcaneum forwards. Look for a sulcus forming in the anterolateral joint line as a vacuum is created in the joint by the subluxing talus. Drawer test is positive if there is more than 4 mm translation. The tilt test can be done with the ankle in neutral. In many people it is possible to hold the talus and tilt it directly while holding the tibia. This allows you to be confident that any tilt is occurring in the ankle. In other patients it is necessary to tilt the heel while holding the tibia (inversion/eversion stress test). A finger on the talar neck will then give an impression of talar movement. Instability of the syndesmosis may be palpable, usually on A-P translation of the distal fibula or valgus stress of the ankle. Abducting the talus or squeezing the tibia and fibula together (the squeeze test) may produce pain from an injured syndesmosis.

Irritability: When you move the joint, does it reproduce the patient's symptoms? This is a useful guide to know whether the symptoms are coming from the ankle. If you suspect the symptoms are coming from the syndesmosis the squeeze test can be useful.

SUBTALAR JOINT

Observe: The shape of the hindfoot and its flexibility as described under general examination. Look for swelling, especially synovitis in the sinus tarsi and the broadening of the hindfoot that occurs after a calcaneal fracture. Look for scars and sinuses.

Palpation: Compare the warmth of the lateral hindfoot with the opposite side. Palpate over each facet for tenderness, bony prominence and synovitis. Palpate the sinus tarsi.

Range of movement: Hold the talar neck and ask the patient to move the heel from side to side. This should give you a rough idea of how much active motion occurs in the free position. Repeat using a hand on the heel to move the joint. A hypermobile joint is often associated with generalized joint laxity; a stiff joint should suggest inflammatory, post-traumatic or degenerative arthritis, post-traumatic arthrofibrosis or tarsal coalition. Pain in the sinus tarsi area maximal on varus tilt is usually due to talocalcaneal ligament injury; pain maximal on valgus stress is usually due to impingement in the calcaneofibular recess after calcaneal fracture, or in the sinus tarsi due to hindfoot valgus with or without inflammatory joint disease.

Stability: The anterior draw or tilt tests holding the talar neck and manipulating the heel may occasionally give a feeling of subtalar laxity, but instability is difficult to demonstrate convincingly even on stress views or arthroscopy.

Irritability: When you move the joint, does it reproduce the patient's symptoms? This is a useful guide to whether the symptoms are coming from the subtalar joint. The injection of local anaesthetic into the joint can also be helpful if it relieves the symptoms.

Sinus tarsi **(Fig. 13.11)**: Remember to examine the sinus tarsi carefully. The "sinus tarsi syndrome" of sinus tarsi pain and tenderness relieved by local anesthetic injection with subjective hindfoot

Fig.13.11: Palpation of tarsal sinus

instability is usually caused by injury to the interosseous talocalcaneal ligament (which may be torn, impinging in the subtalar joint, chronically inflamed or fibrosed) or the subtalar joint arthritis.

Ankle: Remember that many patients with subtalar problems, especially after trauma, have problems with the ankle too, most commonly instability or anterolateral synovitis, so examine the ankle as well.

MIDTARSAL JOINT

Observation: Look for midfoot deformity, swelling and osteophytes from the joint.

Palpation: Compare the warmth of the midfoot with the opposite side. Palpate over the talonavicular and calcaneocuboid joints for tenderness, bony prominence and synovitis.

Range of movement: Hold the heel and ask the patient to move the foot from side to side and up and down (it varies from patient to patient). Repeat the process holding the heel and moving the midfoot to estimate range of midtarsal movement. Adduction is 20° and abduction is 10°.

Stability: The talonavicular joint may be unstable in the flat foot but this is multi-directional and not generally palpable. The navicular drop test gives an estimate of talonavicular instability. Vertical calcaneocuboid instability is occasionally seen, usually with post-traumatic lateral foot pain.

Irritability: When you move the joint, does it reproduce the patient's symptoms? This is a useful guide to whether the symptoms are coming from the subtalar joint. The injection of local anaesthetic into the joint can also be helpful if it relieves the symptoms.

Other structures: If midfoot pain does not appear to be coming from the midtarsal joint, carefully examine the ankle, subtalar and tarsometatarsal joints, the tibialis posterior and peroneus longus tendons and the plantar fascia.

TARSOMETATARSAL JOINTS

Observation: Look for midfoot deformity, swelling and osteophytes from the joint.

Palpation: Compare the warmth of the tarsometatarsal region with the opposite side. Palpate over the tarsometatarsal joints for tenderness, bony prominence and synovitis. Osteophytes dorsal to the first TMTJ are usually innocuous but may indicate instability or arthritis.

Range of movement: Active movement at the TMTJs is almost always zero. Hold the midfoot and manipulate each metatarsal up and down to estimate passive range of movement. Also manipulate the first metatarsal in a valgus-varus plane. When manipulating the first metatarsal, be sure to hold the medial cuneiform in the other hand—the first ray is quite mobile in some people but often most of this movement is in the talonavicular or, usually, the naviculocuneiform joint.

Stability: Stressing the TMTJs may give an impression of instability but this is rare.

Irritability: When you move the joint, does it reproduce the patient's symptoms? This is a useful guide to whether the symptoms are coming from the TMTJ. The injection of local anaesthetic into the joint can also be helpful if it relieves the symptoms.

Other structures: If midfoot pain does not appear to be coming from the tarsometatarsal joints, carefully examine the subtalar and midtarsal joints, the tibialis anterior and posterior and peroneus longus tendons and the plantar fascia. The planovalgus foot may have some laxity at the tarsometatarsal level and a vague midfoot ache, which is not reproduced by TMTJ manipulation or blocked by local injection.

ACHILLES TENDON

History: As well as the general questionnaire, establish in detail the patient's level of sporting

activity and whether anything has changed recently—distance, running surface, shoes. Always ask not only about trauma but non-traumatic acute pain in the tendon. Acute pain during sport or other vigorous activity followed by swelling and a limp suggests a torn Achilles tendon.

Observation: Lack of push-off in the propulsive stage of gait suggests Achilles weakness or rupture. A tendency to walk with the foot in valgus may be due to a tight Achilles tendon. Inability or difficulty with walking on the heels also suggests a tight tendon.

Palpation: Feel the gastrocnemius and soleus bellies and the whole length of the tendon, feeling for gaps, tenderness, swelling, or paratendonitis. Distinguish between paratendonitis, in which there is generalised tenderness and puffiness with a slightly crinkly feeling, insertional tendonitis in which the pain and swelling is at the insertion of the tendon **(Fig. 13.12)** and there is often a Haglund's prominence, and non-insertional tendonitis in which the swelling is in the substance of the tendon about 3 to 6 cm above the insertion. Also identify the posterolateral (Haglund's) prominence of the calcaneum and palpate the retro-Achilles bursa.

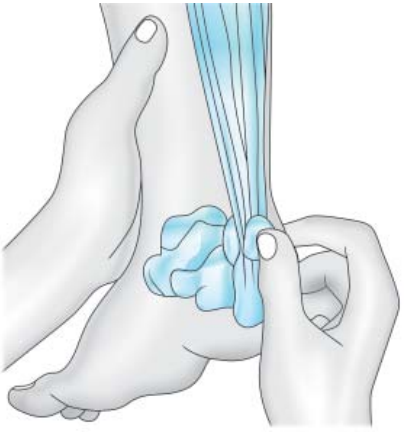


Fig. 13.12: Palpation of Achilles tendonitis and retrocalcaneal bursitis

Integrity: Some ruptured tendons have an obvious gap. The best test for integrity of the tendon is the Thompson (or Simmond's) test: squeezing the calf produces plantar flexion of the foot equal to that on the normal side. Do not be misled by the ability to stand on tiptoe—it does not exclude an Achilles tendon rupture.

Strength: Compare active and resisted plantar flexion with the opposite side but remember this does not only test the triceps surae. If weak, check that the Achilles tendon is intact and examine the S1 root sensory supply.

Contracture: Assess the range of passive ankle dorsiflexion with the heel in the neutral position (this is particularly important in planovalgus feet in which the heel tends to go into valgus when the ankle is dorsiflexed giving a spurious impression of no Achilles contracture). If restriction of dorsiflexion is greater with the knee extended than flexed the contracture is principally in the gastrocnemius, whereas restriction, which is equal in all knee positions, is due to the soleus. Remember that there are other causes of restricted ankle dorsiflexion: Capsular contracture or arthrofibrosis of the ankle after trauma, anterior ankle impingement, ankle arthritis and other soft tissue contractures.

TIBIALIS POSTERIOR

History: The classic complaints in tibialis posterior problems are posteromedial ankle pain and swelling and gradual (occasionally sudden) arch collapse. Many people with tibialis posterior insufficiency have a pre-existing flat foot.

Observation: There may be swelling along the course of the tendon, especially behind and below the medial malleolus. The foot may be flat. Examination from behind may show a valgus heel, prominent talar head and the "too many toes sign" indicating an abducted forefoot. The shoes will show pressure and buckling if there is a significant flat foot.

Palpation: Palpate the entire muscle and tendon, looking for swelling, gaps, tenderness and synovitis.

Integrity: From behind, ask the patient to do a single foot tiptoe test on both sides. Most people cannot get the affected heel off the ground at all; a few develop an acute midfoot breach. Another useful test is to get the patient to contract the tibialis posterior in the plantar-flexed/inverted position **(Fig. 13.13)**. The tendon may be weak, impalpable or palpably thin. The plantar flexed position prevents recruitment of the tibialis anterior. Always examine for an Achilles contracture, which is present in most people with tibialis posterior insufficiency.

Strength: Strength can be tested both actively and against resistance in the plantar-flexed/inverted position. Always test the strength of the other muscles too.

Contracture: A fixed tibialis posterior contracture presents with a fixed equinovarus foot. After a stroke tightness or inappropriate firing of the muscle may present with a dynamic equinovarus that may only be clear on formal gait analysis. The Achilles tendon is usually tight too.

DORSIFLEXORS

History: Tendonitis of the dorsiflexors is uncommon and usually presents in athletes. Foot drop may present after stroke, spinal injury, stenosis or disc prolapse, peripheral nerve injury or neuropathy, requiring a thorough examination and often further investigation.

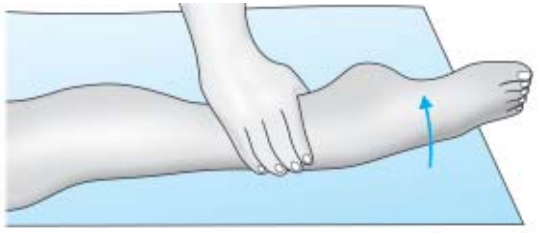


Fig. 13.13: To test tibialis posterior have the patient to supinate and invert the foot from a lateral position

Observation: The gait of foot drop is the classical high step and flop. Tendonitis of the dorsiflexors causes pain and affects gait in the early contact phase, especially when going uphill. The dorsiflexors are often weak in pes cavus, but are rarely the cause of complaint in this condition.

Palpation: Palpate the entire muscles and tendons, looking for swelling, gaps, tenderness and synovitis. Feel for synovium protruding between the limbs of the inferior extensor retinaculum.

Integrity: Resisted dorsiflexion with palpation of the tendons should assess tendon integrity.

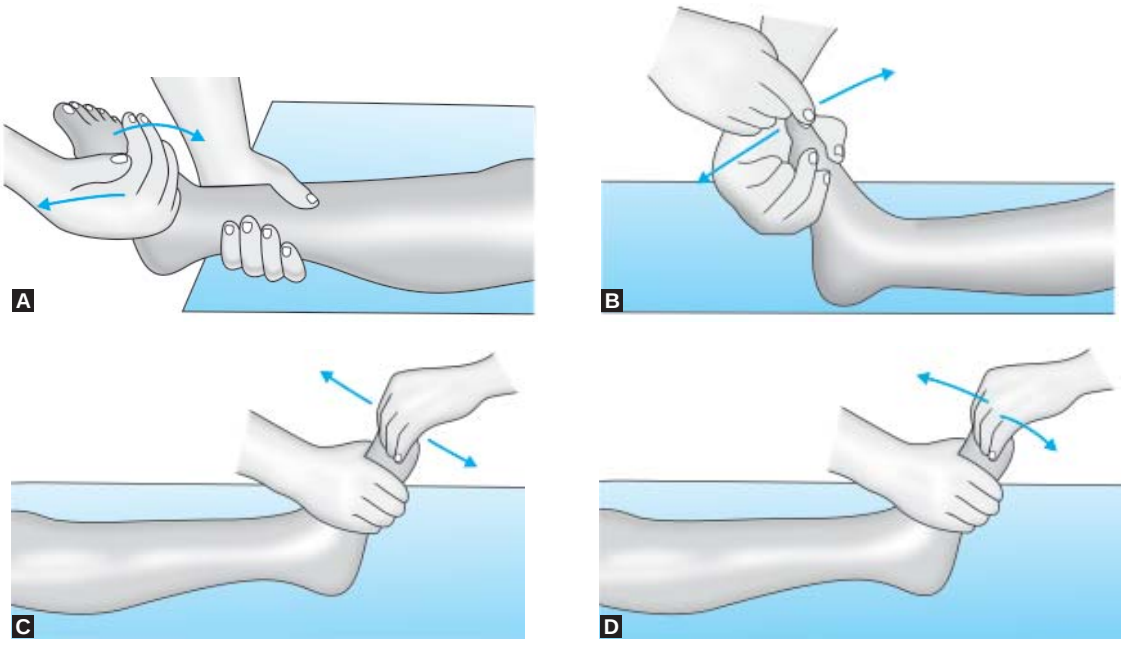
Strength **(Figs 13.14A to D)**: Active and resisted dorsiflexion will allow estimation of strength. Weakness should lead to evaluation of the CNS, L5 spinal level and the sciatic, common and deep peroneal nerves.

Contracture: Dorsiflexor contracture is rare, except where the toe metatarsophalangeal joints are pulled into dorsiflexion by muscle imbalance and loss of passive toe control.

PERONEALS

History: Peroneal tendon problems present with lateral hindfoot pain or pain under the midfoot. It may be precisely located to the point where the peroneus longus curves under the foot (sometimes known inaccurately as POPS—painful os peroneum syndrome). Another presentation is with pain, swelling and sometimes popping or obvious dislocation of the tendons behind the lateral malleolus. Patients with peroneal instability may complain that the ankle gives way.

Observation: The gait, if affected, tends to be antalgic. There may be swelling over the lateral hindfoot. Occasionally the peroneal tendons are obviously dislocated from behind the malleolus or can be made to do so by the patient.



Figs 13.14A to D: (A) Tibialis anterior is tested by stabilizing the calf above the ankle with one hand while exerting pressure in plantar flexion and eversion on the dorsum of the foot with the other hand against resistance; (B) Extensor hallucis longus is tested by immobiling the tarsus with one hand and apply dorsal pressure to the distal phalanx of great toe with the examiner's other hand against resistance; (C) Extensor digitorum longus is tested by immobilizing the tarsus and applying dorsolateral pressure to the distal small toes; (D) Extensor digitorum brevis is tested by immobilizing the tarsus and applying dorsolateral pressure to the proximal phalanges of small toes

Palpation: Palpate the entire muscles and tendons, looking for swelling, gaps, tenderness and synovitis. Post-malleolar tendonitis may produce a "popping" or grating sensation on compression. It may be possible to dislocate the peroneal tendons, especially with the foot in dorsiflexion/eversion. Carefully palpate around the peroneal trochlea and the peroneus longus where it goes under the foot as these are also common sites of tendon problems.

Integrity: Testing of eversion, both active and resisted, gives an estimate of peroneal integrity, but major tendon defects may be present without palpable weakness. Inability to plantar flex the first metatarsal is typical of peroneus longus rupture but is difficult to test and FHL can compensate. Test the integrity of the peroneal retinaculum by attempting to dislocate the tendons with the ankle dorsiflexed and the foot in eversion.

Strength **(Fig. 13.15)**: Testing of eversion, both active and resisted, gives an estimate of peroneal strength.

Contracture: Peroneal contracture is not often seen but produces fixed hindfoot eversion and first ray plantarflexion. "Peroneal spasm" is said to be a feature of tarsal coalition but the muscles are usually contracted secondary to the hindfoot valgus rather than truly in spasm.

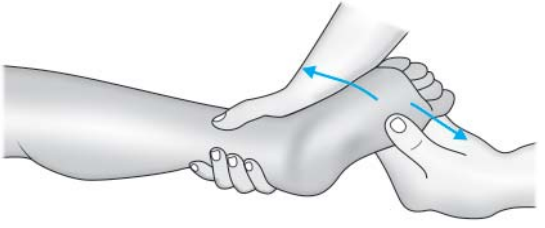


Fig. 13.15: Peroneus longus and brevis is tested by keeping the leg internally rotated, immobilizing the calf slightly proximally to the ankle with one hand while pressing the lateral margin of the foot into adduction and inversion with the other hand against resistance

HEEL

History: Heel pain is usually due to plantar fascitis but several other conditions can produce this complaint. Ask particularly about trauma, diabetes, spinal problems, arthritis and other features of inflammatory disease, symptoms suggestive of nerve entrapment such as numbness, burning and electric shock sensations. Clarify exactly where the pain is felt: many patients with plantar fascitis point directly to the medial calcaneal tubercle **(Fig. 13.16)**, whereas in heel pad atrophy the whole heel pad is sore, and other patients will indicate the medial, lateral or posterior aspect of the heel, not the underside, as the source of pain. Ask about the diurnal rhythm of the pain: plantar fascitis is typically worse on getting out of bed and then gets worse towards evening, while heel pad atrophy tends to be simply activity related. Ask about occupation: heel pain is more common in those who are on their feet a lot, especially if they work on a hard surface in hard shoewear, and obviously this may affect the ability to return to work.

Observation: The early part of the stance phase of gait is shortened as the patient unloads the painful heel. Obesity is common in patients with plantar heel pain. Look for other evidence of inflammatory arthritis: joint deformity, psoriasis, rheumatoid nodules. Look in the shoes: often patients have tried homemade or proprietary heel pads, which are often of little benefit.

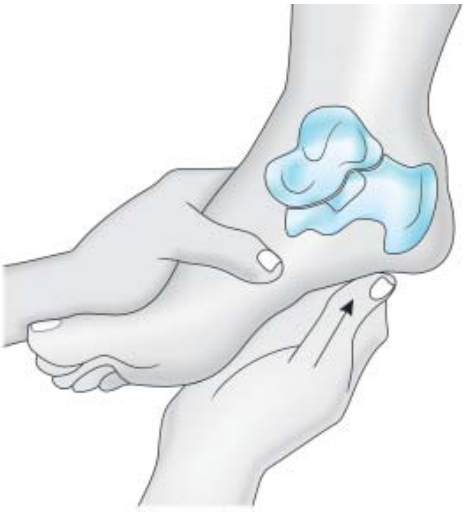


Fig. 13.16: Palpation of heel spur

Palpation: Palpate all round the heel, checking the medial calcaneal tubercle, the rest of the undersurface, the medial hindfoot (especially the nerves in the tarsal tunnel and the medial side of the calcaneum and the nerve to abductor digit quinti where it passes under the heel), the Achilles tendon insertion, the peroneal and tibialis posterior tendons and the ankle and subtalar joints, manipulating the latter to see if they are unduly stiff or irritable. Always examine for an Achilles tendon contracture, which is often present in patients with plantar fascitis.

THE GREAT TOE

History: Decide exactly what the patient is complaining of: Cosmetic dissatisfaction, medial eminence pain, dorsal MTPJ pain, joint pain, stiffness, shoe problems, other pressure problems such as kissing corns between the great and second toes. Make a realistic assessment of the patient's attitude to shoes and their willingness to look for shoes that fit the foot. Always ask about lesser toe problems, lesser metatarsalgia and generalized arthropathies.

Observation: With a stiff or severely valgus great toe the propulsive phase of gait may be weak or even absent. The shoes will show deformation by a substantial bunion or dorsal exostosis. Assess the severity of the hallux and any lesser toe deformities in the standing position. Look for skin breakdown or sinuses over bony prominences. Distinguish between a medial prominence (hallux valgus) and a dorsal exostosis (hallux rigidus). Look for sub-metatarsal calluses. Interspace fullness can be due to synovial pathology, neuroma or bursitis.

Palpation: Palpate for tenderness, swelling, synovitis and joint instability. Especially localize tenderness around the hallux **(Fig. 13.17)**. Medial pain can be due to exostosis, dorsomedial cutaneous nerve irritation or bursitis/synovitis especially in gout. Sesamoid arthrosis can cause pain in the plantar aspect. Feel for tenderness under the lesser metatarsal heads.

Movement: Assess the range of movement in the hallux MTP and IP joints, the lesser toes and the rest of the foot, both passively and actively. If there is hallux valgus, test the MTP joint in the corrected position if possible. Assess how correctable the hallux valgus is. If there is hallux rigidus, measure how much movement remains, in both plantar and dorsiflexion. Manipulate the hallux MTP joint for irritability in both the neutral (arthritic pain) and dorsiflexed (dorsal impingement pain) positions. Hallux valgus interphalangeus is usually most apparent with the IP joint flexed, although with practice it can usually be diagnosed with the toe straight. Assess the range of movement of the proximal first ray in both sagittal and transverse planes. Test the lesser MTP joints, especially the second, for instability. All toes are tested for strength **(Figs 13.18A to C)**.

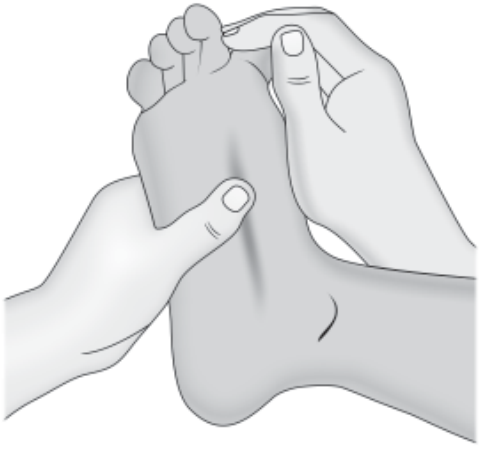


Fig. 13.17: Palpation of flexor hallucis longus tendon

GREAT TOENAIL AND NAIL BED PROBLEMS

History: Which part of the nail is the patient complaining of? If the problem is recurrent, is there an obvious reason, such as diabetes or systemic steroid use? What has been done in the past? Take a careful history of lesions under the nails - remember some of them are tumors. If the nails are dystrophic, a general medical history should be taken to look for the cause.

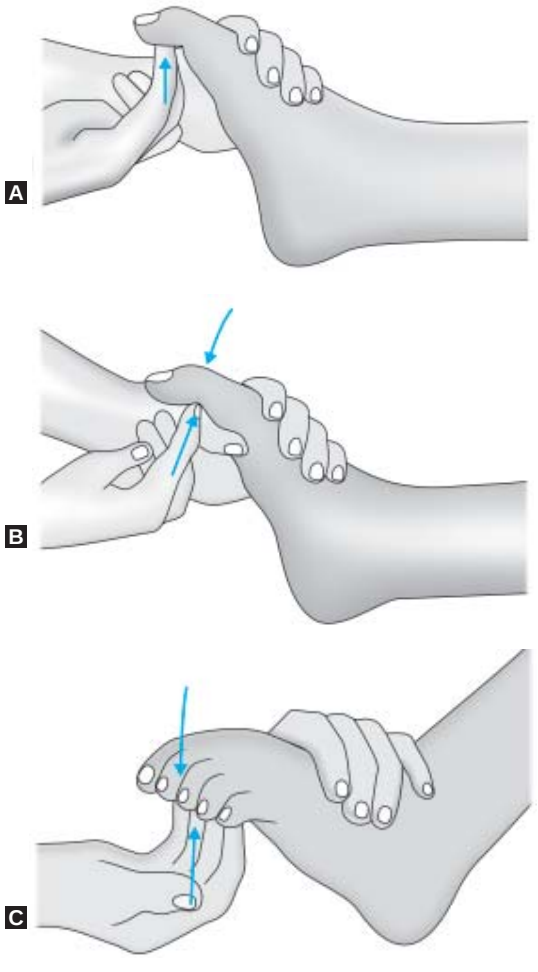
Observation: Look for nail deformity and dystrophy. Are one or both nail folds inflamed? Is there any spreading cellulitis or abscess formation? Look for lesions under the nail. Remember that melanomas are not always pigmented.

Palpation: If there is active infection or a subungual lesion palpate the regional lymph nodes.

METATARSAL REGION

History: Patients generally complain of pain, which we usually call "metatarsalgia." Sometimes this term is used more specifically of pain under the metatarsal heads. There are many possible causes, not all in the foot. Always remember to think about:

a. Obesity
b. Diabetes



Figs 13.18A to C: (A) Flexor hallucis longus is tested by applying pressure to the distal phalanx of the reat toe as shown against resistance; (B) Flexor hallucis brevis is tested by applying pressure to the proximal phalanx of the great toe as shown against resistance; (C) Flexor digitorum longus is tested by applying pressure to distal phalanges of small toes as shown

c. Generalized arthropathy such as rheumatoid disease
d. Biomechanical problems such as a tight Achilles tendon, short or dysfunctioned first ray
e. Lesser toe deformities
f. Neurological problems such as interdigital neuralgia or, more rarely, tarsal tunnel syndrome.

Other people complain only of calluses under the forefoot (usually pressure-related) or tingling, numbness or other neurological symptoms, which are usually due to interdigital neuralgia.

Observation: The gait may be antalgic or there may be a poor propulsive phase. Examination of the spine, proximal limb, leg lengths or other joints may be necessary and a more or less full neurological examination is often needed. Carry out a full examination of the proximal foot as detailed in previous sections, particularly looking at the overall shape of the foot and for evidence of arthritis, trauma and previous surgery.

Look at the forefoot, again looking for deformities of the overall forefoot and toes, arthritis/synovitis, scars, swellings, malalignments. Assess toe deformities with the patient standing. Look carefully for corns and other skin lesions between the toes. Look carefully for calluses under the metatarsal heads— Do they correspond with the patient's complaints?

Palpation: Again, palpate the proximal foot as detailed in the sections above. Palpate the forefoot, feeling for tenderness, swelling, and malalignment. Differentiate between tenderness in the MTP joints, under the metatarsal heads and between the metatarsal heads. Test for a Mulder's click. Examine the pulses, using the Doppler if necessary, and examine the foot neurologically including pressure testing with Semmes-Weinstein hairs if diabetes is present or suspected.

Movement and stability of joints: Assess active and passive range of movement of MTP and interphalangeal joints. Evaluate the stability of all the MTP joints and the reducibility of lesser toe deformities in plantar flexion. How stable overall is the first ray?

Injections: Diagnostic injections of local anesthetic can be very useful in locating the source of

forefoot pain. Usually inject joints first before testing nerves.

LESSER TOES

History: Complaints usually relate to toes, which rub on the shoes (usually over a PIP joint or at the tip) or on each other (usually with a bony prominence on a condyle underneath). Some people are unhappy with the appearance of their toes. Some lesser toe problems present with metatarsalgia. Multiple toe problems, especially if the toes look odd or there is pes cavus or muscle wasting, may be associated with a generalized neurological disorder or a spinal malformation. Interdigital neuralgia sometimes presents with symptoms mainly in the toes.

Observation: Sometimes the gait is antalgic or there is a poor propulsive phase. Look at the entire lower limbs for muscle wasting or other deformities, which may suggest a neurological or malformation syndrome. Look at overall foot shape, especially pes cavus or a severely overpronated foot. Look for evidence of generalized arthropathy or vascular insufficiency. Assess the shape of the toes with the patient standing. Look for calluses under the metatarsal heads, over the PIP joints and at the tips of the toes, and soft interdigital corns and other skin lesions.

Palpation: Palpate the proximal foot as detailed in the sections above. Palpate the forefoot, feeling for tenderness, swelling, malalignment. Palpate the toes for tenderness, swellings and synovitis. Feel for any soft corns carefully underlying bony prominences. If there are symptoms suggestive of interdigital neuralgia, feel for a Mulder's click with two fingers of one hand gently palpating the interdigital space while the other hand compresses the metatarsal heads together. Examine the pulses, using the Doppler if necessary, and examine the foot neurologically including pressure testing with Semmes-Weinstein hairs if diabetes is present or suspected.

Movement and stability of joints: Assess active and passive range of movement of MTP and interphalangeal joints. Evaluate the stability of all the MTP joints and the reducibility of lesser toe deformities and estabilised contractures.

SPECIFIC CONDITIONS

Pes Cavus

Clinically cavus can be made out by high medial arch and lateral aspect of foot not touching the ground that can be tested by passing a coin.

Coleman's Block Test (Figs 13.19A and B)

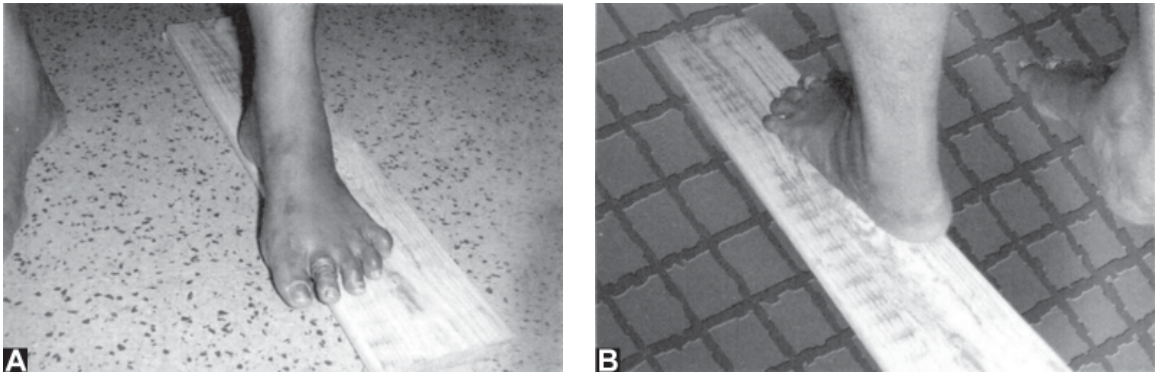
To understand this test, it is essential to know the mechanism of pes cavus. Usually it starts due to muscle imbalance resulting in dropping of 1st metatarsal, this creates a pronation deformity of the foot. To make the foot flat on the floor the hind foot inverts. In this special test a wooden block is kept under the heel and lateral rays so that the 1st ray is allowed to drop freely. This results in reproduction of forefoot pronation with the hind foot in neutral position. If the hind foot varus does not correct with this test, then it indicates fixed deformity of subtalar joint due to long-standing deformity. This test essentially differentiates flexible from rigid hind foot varus deformity.

Look for clawing of hands, which may indicate the diagnosis of Charcot-Marie-Tooth disease.

Assess spine for tethered cord syndrome or any other spinal disorders.

Check for Romberg sign, which is failure to keep the body balance upright on closing the eyes. This indicates posterior column lesion and favors the diagnosis of Friedreich's ataxia. Diseases causing pes cavus includes:

i. Neuromuscular cause Friedreich's ataxia, Charcot-Marie-Tooth disease, cerebral palsy, poliomyelitis, spinal dysraphism
ii. Post-traumatic malunited fractures, compartment syndrome, crush injury foot.



Figs 13.19A and B: Coleman's block test

iii. Inflammatory condition rheumatoid arthritis
iv. Congenital residual club foot, arthrogryposis, idiopathic.

Flat-foot

Look for too many toes sign from the back in standing posture **(Fig. 13.20)**.

Flexible or Rigid Flat Feet

In flexible flat feet the medial arch is present on non-weightbearing and disappears on weight bearing. On standing on tiptoes the arch reappears in flexible flatfeet. This can also be checked by Jack test in which dorsiflexion of big toe reproduces the medial longitudinal arch.

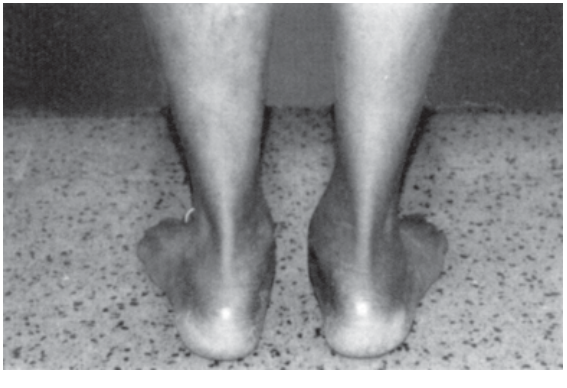


Fig. 13.20: Too many toes sign

Tibialis Posterior Insufficiency Test

This can be tested by 'single leg stance' on tiptoes, which produces inversion of hind foot due to intact tibialis posterior. Patient with rupture will not be able to stand on one leg on tiptoes. The muscle can also be assessed for contraction by bending the knee, with foot in equinus and performing resistant inversion. Always feel for taut tendon 2 cm behind and above the medial malleolus. Plantarflexing the foot eliminates the action of tibialis anterior as invertor.

Tight Tendo-Achilles

This should be tested by bringing the valgus heel into neutral position and dorsiflexing the foot to reveal tightness. It is a cause for flexible flatfoot due to ligamentous laxity.

Conditions causing flatfeet include:

Congenital: Flexible—tight heel cord

Rigid—tarsal coalition, vertical talus, arthrogryposis

Acquired: Tibialis posterior insufficiency

Rheumatoid arthritis

Diabetes

Degenerative joint disease of tarsometatarsal joint

Traumatic-calcaneal fractures

Neuromuscular—polio, cerebral palsy, nerve injuries

Club Foot (Congenital Talipes Equino Varus

It is a congenital deformity of the foot characterized by stork-like legs, equinus of ankle, inversion at subtalar joint and adduction of forefoot. It is bilateral in 50 percent and 90 percent is idiopathic in origin **(Fig. 13.21)**.

Size of the foot and leg is always comparatively smaller, with deep medial crease, convex lateral border and raised posterior heel. Postural club foot is differentiated from true club foot by dorsiflexing the foot and in which case the dorsum can touch the shin of tibia. Presence of very short 1st ray and absent posterior skin crease over calcaneum with small heel indicates rigidity of the foot. It can be differentiated from neurogenic clubfoot by stroking the sole, which should normally cause dorsiflexion of the foot. Examine the ipsilateral hip for developmental dislocation of hip and spine for any obvious neurocutaneous markers. A child with a congenital anomaly should be screened completely for other associated anomalies. It is important to know the prenatal, perinatal and postnatal history, nature of birth, any family history of similar problems and delayed milestones. Child needs opinion from a pediatrician and the family needs genetic counseling. It can be associated with multiple congenital contractures of the joint, otherwise called arthrogryposis multiplex congenita **(Fig. 13.22)** which is a nonprogressive disorder with multiple congenital rigid joints due to disorder of myopathic, neuropathic (decreased in anterior horn cell) or mixed affection. They have normal intelligence, absence of shoulder muscles, thin tubular limbs, elbow extended, clasp thumb, wrist flexed, no flexion creases, teratologic hip dislocations, knee contractures, resistant club feet and vertical talus.

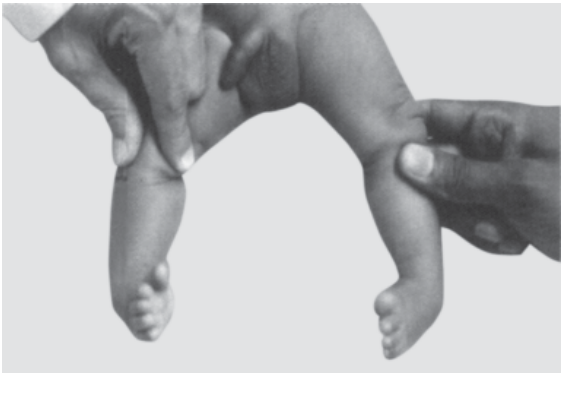


Fig. 13.21: Bilateral club foot

Plantar Fascitis

Heel pain with tenderness in plantar medial aspect of calcaneum. This can be due to inflammatory diseases, sudden increase in weight, plantar flexed 1st ray on forefoot valgus. This condition is treated by anti-inflammatory drugs, modified footwear, local heat treatment and sometimes night splint in dorsiflexion. Presence of calcaneal spur rarely causes pain. Differential diagnosis includes neurologic causes—lumbar disk prolapse, tarsal tunnel syndrome, and entrapment of posterior tibial nerve branches; stress fracture of calcaneus and heel pad atrophy.

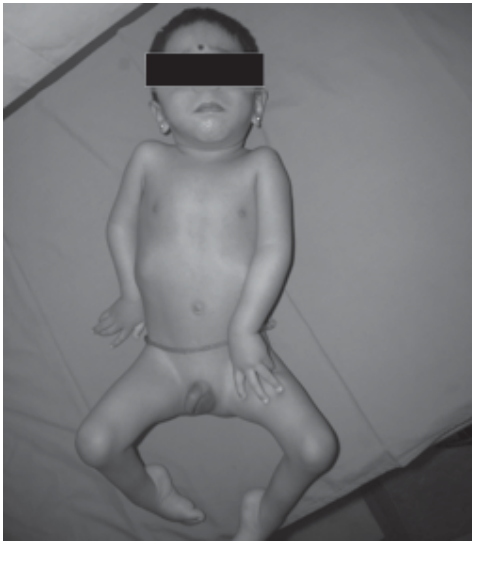


Fig. 13.22: Arthrogryposis multiplex congenita
(For color version, see Plate 7)

Posterior Heel Pain

It can be from insertional Achilles tendonitis, retrocalcaneal bursitis, and Haglund's disease.

Tendo-Achilles Rupture

This condition presents with sudden giving way and pain at the heel cord region, sometimes with a feeling of being tripped by somebody from back (in attrition rupture). Patient feels the leg is weak with loss of push off and is unable to stand on tiptoes on involved side. There may be swelling or ecchymosis in the heel cord area. A gap may be felt in complete rupture (commonly about 2 inches above its insertion) with abnormal range of dorsiflexion. This is confirmed by Simmond-Thomson's test.

Simmond-Thomson's Test (Fig. 13.23)

The calf is squeezed while the patient lies prone with the foot projecting beyond the edge of the couch or by kneeling on the chair. Plantarflexion of foot is seen if the tendo-Achilles tendon is intact or incompletely ruptured. In complete rupture of tendon there will be no plantar flexion of the foot.

Osteochondritis of Talus (Osteochondral Dome Fracture or Partial Talar Necrosis)

Patient presents with pain, giving way of ankle, and occasional swelling.

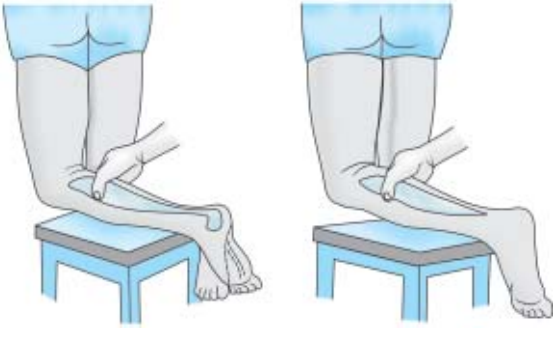


Fig. 13.23: Thomson's test

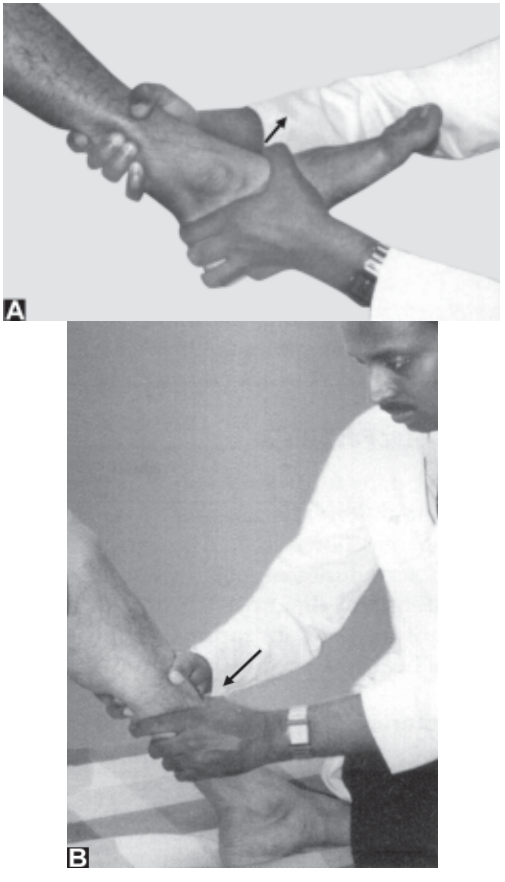
Osteochondritis of Navicular (Avascular Necrosis of Navicular Bone)

It is also called Kohler's disease. It is common in children who present with midfoot pain and is a self-limiting condition.

Ankle Instability

Anterior Drawer Test (Figs 13.24A and B)

It can be done by sitting on the foot and pushing the shin of the tibia back or holding the talus in equinus in one hand and the other hand to stabilize the lower leg, perform anterior drawer test. This implies injury to lateral ligament complex.



Figs 13.24A and B: Anterior drawer test

Inversion-Eversion Stress Test

This can be assessed clinically and radiologically by talar tilt. Isolated anterior talofibular ligament injury is tested by inversion of the foot in equinus position. Inverting the hind foot in neutral position tests calcaneo-fibular ligament and any opening in neutral position indicates ankle instability and subtalar joint instability. Talar tilting of more than 5° in comparison to opposite side can be pathological.

Osteoarthritis Ankle

It can be primary or secondary and patients present with pain on walking and stiffness. Swelling of the ankle can be due to bony outgrowth-osteophytes, soft tissue synovial thickening or effusion of the joint. As the ankle joint gets stiffer it lacks the normal rocking of the gait cycle. Patients try to externally rotate the foot to shorten the lever arm of the dorsiflexion and plantarflexion movement.

Subtalar Arthritis

It can be of infective, post-traumatic or inflammatory cause. Patients present with difficulty in walking on uneven surface and morning stiffness. Some patients can have peroneal spasm. Inversion and eversion movements may be restricted.

Metatarsalgia

Foot and toes deformities should be assessed. Plantar callosities should be identified and felt for tender spot both on metatarsal heads and intermetatarsal space. Spine should be examined if the foot has pes cavus with clawing of toes. Any previous surgery should be noted especially hallux valgus correction by 1st metatarsal osteotomy that results in shortening of the ray and transfer metatarsalgia to lesser rays. Inflammatory conditions, Freiberg's infraction (osteochondritis of 2nd MT head), tibial tunnel syndrome, viral warts, stress fractures, tumours and infections are some of the causes of metatarsalgia. This can be investigated by pedobarography to assess the areas of weightbearing. Appropriate insoles by distributing the weight evenly under the foot can solve this problem.

Morton's Neuroma

Usually it is a neuroma of the 3rd digital branch of medial plantar nerve, manifests with metatarsalgia and sometimes numbness and tingling between 3rd and 4th toes. Patients present with pain on walking, well-localized and patients wearing shoes note that taking the shoes off relieves the pain. Some patients feel sitting crosslegged causing pain. Mulder's click may be present **(Fig. 13.25)**. This is clicking sensation on squeezing the forefoot like a spring test. Tenderness in the intermetatarsal space **(Fig. 13.26)** and sometimes a palpable nodule can be found. Examine the toes for neurological deficit. In doubtful cases either a trial injection can be done or a MRI scan to confirm the diagnosis.

Metatarsus Adductus

Characterized by adduction of forefoot and commonly associated with developmental dysplasia of hip. Bleck described a grading

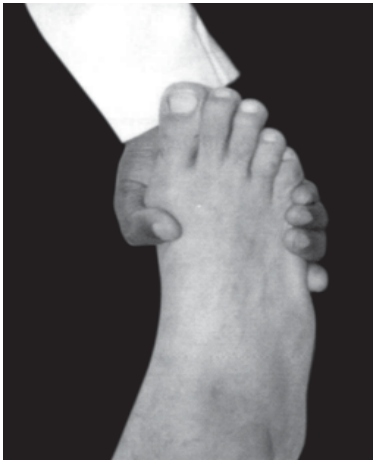


Fig. 13.25: Mulder's click

system based on heel bisector line, which normally passes through second and third toe interspace **(Fig. 13.27)**.

Hallux Valgus (Fig. 13.28)

Outward deviation of big toe at Ist metatarsophalangeal joint. Assess the length of the 1st ray in comparison to 2nd ray, presence of medial bunion, plantar callosities to know the weightbearing part in the sole, and range of movement of the joint. Pain on movement indicates inflammation or arthritis. Attempt to passively correct the deformity; this gives an indication on soft tissue tightness. Extreme hallux valgus can result in hammer 2nd toe. Hallux inter-phalangeus can also result in hallux valgus. Metatarsus primus varus should be identified clinically and radiologically, as it needs basal osteotomy for correction. Also examine the 1st tarsometatarsal joint for hypermobility.

Hallux Rigidus

More common in young and middle-age and and is unilateral disease. Presents sometime with dorsal bunion and inability to stand on tip toes. I st MTP joint movement is very much restricted with pain. Weightbearing foot X-ray AP and lateral is essential.

Gout (Figs 13.29A and B)

It presents with signs of acute inflammation of 1st MTP joint. In chronic gout there may be gouty tophi over the pinna of ear and fingertips. It is related to alcohol and food intake. This crystal arthropathy is due to deposition of monosodium urate crystals. Serum uric acid may be elevated.

Tarsal Tunnel Syndrome

Tibial nerve is constricted beneath the flexor retinaculum in the tunnel formed by septae from this fibrous roof to calcaneum. Unexplained

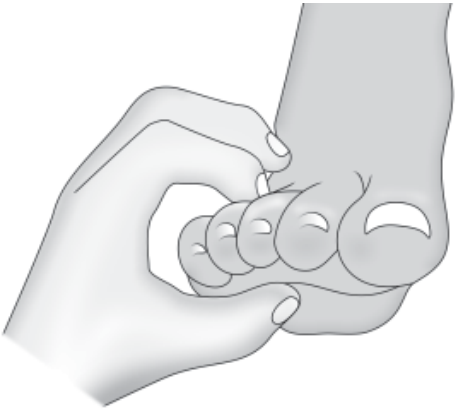


Fig. 13.26: Intermatatarsal tenderness

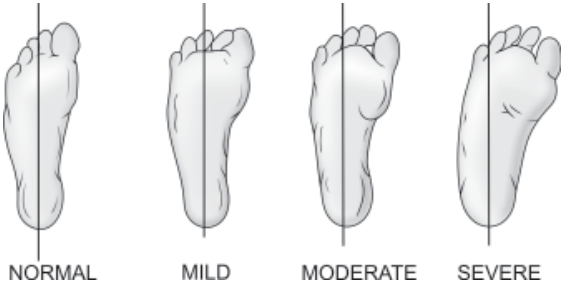


Fig. 13.27: Heel bisector line

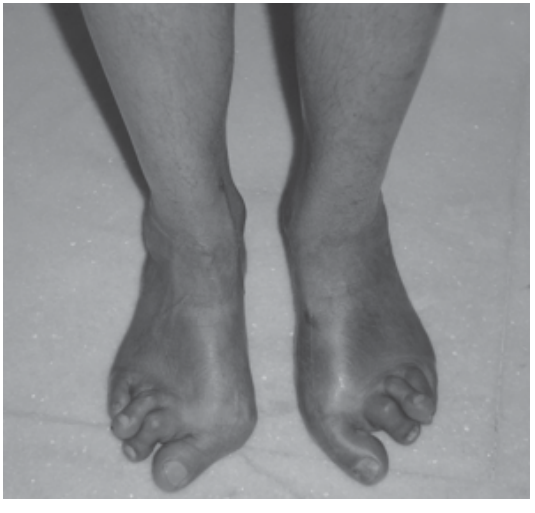


Fig. 13.28: Bilateral hallux valgus
(For color version, see Plate 7)

paresthesia in the plantar aspect of foot and toes aggravated at night or by exertion. This can be due to talar or calcaneal fractures, tenosynovitis, ganglia, rheumatoid synovitis, varicosities etc. This can be confirmed by EMG and nerve conduction test.

Neuropathic Joint (Charcot Arthropathy)

Chronic progressive, destructive process affecting the joint alignment due to lack of proprioception. Extreme form of arthritis typically characterized by unstable, painless, swollen joint with radiographs showing advanced destructive changes of both sides of joint, chunks of bone all around with distortion of the joint. Because of warmth and destruction and new bone formation it is confused with osteomyelitis but patient will not be in acute pain and etiology may be obvious. Commonest cause is diabetes involving foot, syringomyelia involving shoulder and elbow, Hansen's disease involving upper or lower limb joints, spinal cord injury or spinal dysraphism, nerve injuries, congenital insensitivity to pain ant tabes dorsalis are other causes **(Figs 13.30 and 13.31)**.

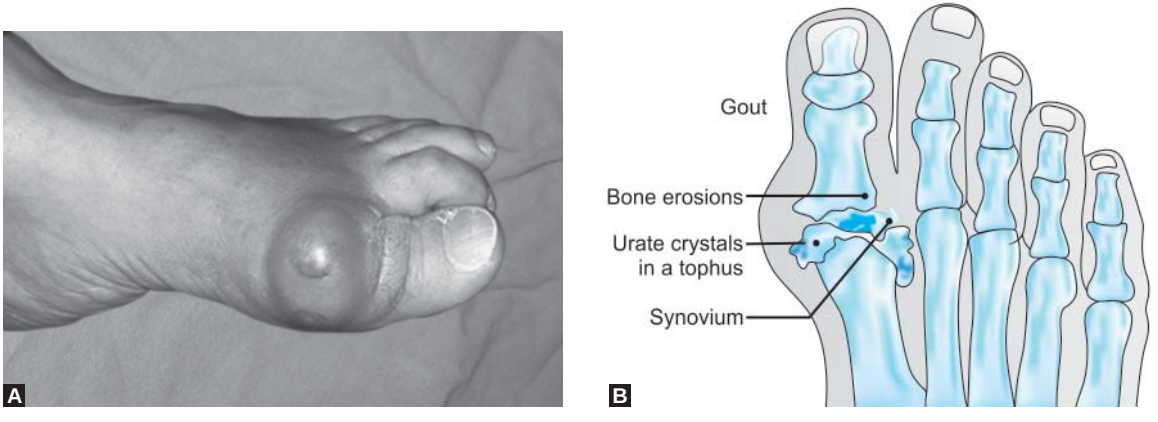


Fig 13.29A and B: (A) First metatarsophalangeal joint acute inflammation from gout; (B) Pathology in gout

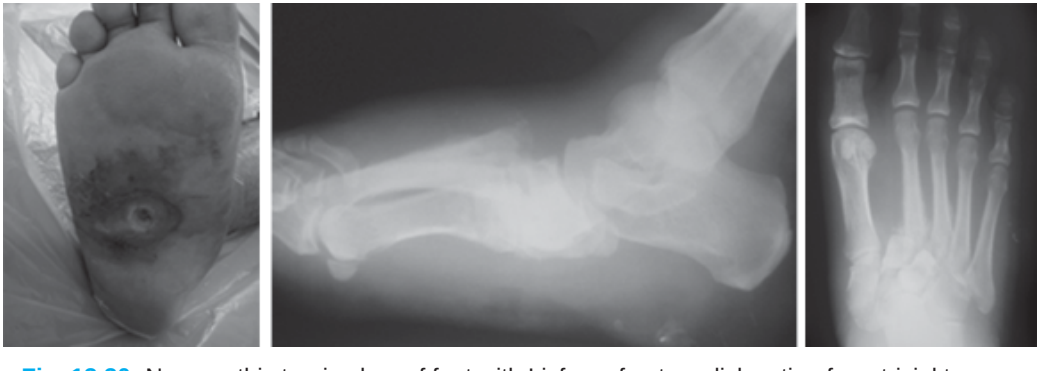
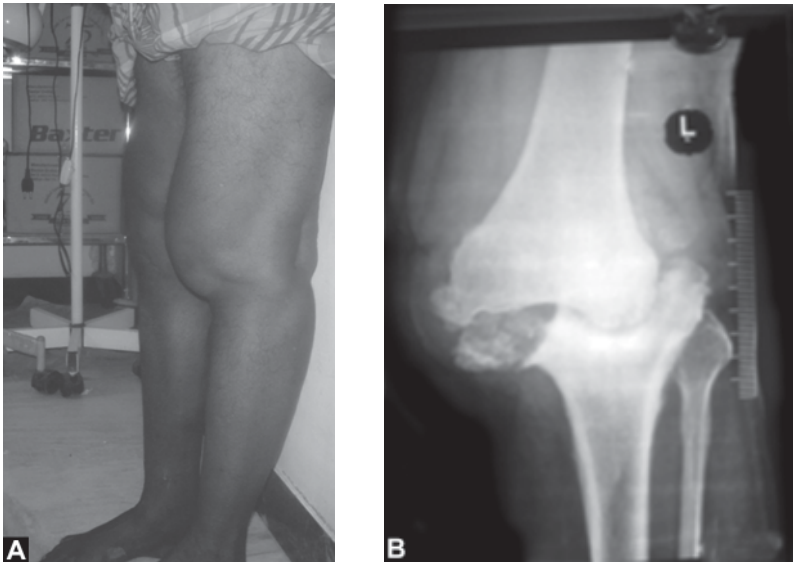


Fig. 13.30: Neuropathic tropic ulcer of foot with Lisfranc fracture dislocation from trivial trauma
(For color version, see Plate 8)



Figs 13.31A and B: Neuropathic knee joint *(For color version, Fig. 13.31A, see Plate 8)*

Neuropathic Foot

This may start with a trivial fracture or injury to foot in a patient with sensory disturbance. The most common cause is diabetes, but can also occur in tabes dorsalis, spina bifida, leprosy, congenital indifference to pain and nerve injuries. Patient can have pain, lot of swelling, increased warmth, deformity, trophic ulcer and bony thickening. There is total disorganization of the tarsal joints with new bone formation. The proportion of pain is less for the damage that has occurred and in some patients it may be totally pain free.

14 CHAPTER Examination of Rotational Deformities in Lower Limb

Clinical evaluation of rotational deformities of lower extremity in children is important. Rotational deformities in children manifest with toeing in or toeing out. The deformity can be in the femur, tibia or foot. The usual presentation is for cosmetic reasons or sometimes with gait disturbance, patellofemoral disease and arthritis.

Version refers to normal rotational alignment of tibia and femur; torsion is when the deformity is abnormal. In common practice version is used for describing femoral rotational alignment and torsion for tibia.

HISTORY

Presentation

The family may complain of: (i) clumsiness, (ii) difficulty in running, (iii) frequent falls and (iv) cosmetic appearance.

Prenatal History

Birth history and milestones must be asked.

Family History

Some of the deformities may run in the family and this must be asked for.

Knowing patient's or parents' expectation

EXAMINATION

Gait can be in-toeing or can have other abnormalities.

Examination on Standing

Assess the normal limb alignment in sagittal plane. The normal plumb line (mechanical axis) is from midinguinal point (midpoint between antero-superior iliac spine and pubic symphysis) through midline of knee joint and passes through second toe. In-turning of patella due to excessive femoral torsion may be associated with toeing in or out depending upon tibial rotation. In early stages of excessive femoral anterior torsion, in toeing is present, but in late stages normal toeing out is present because of compensatory lateral tibial rotation following growth **(Table 14.1)**.

Examination on Sitting

Sitting with legs hanging down, axis of thigh to intermalleolar axis gives an indication of tibial torsion. Normally the tip of lateral malleolus is 1 cm behind and below the medial malleolus. Child with significant anteversion often sits in "W" position that is "both hips fully internally rotated and legs out."

Always check the hip for dysplasias, spine for any abnormalities, knee for patellofemoral malalignment and increased "Q" angle, and foot for deformities.

Assessment of Rotational Profile

Staheli's Rotational Profile Tests

Staheli's rotational profile tests are used to identify the site of rotational deformity **(Fig. 14.1)**.

Table 14.1: Imaging principles

Modality	*Source*	*Result*	*Radiation*	*Vocabulary*	*Indication*
X-ray	X-rays	2-D Shadow	Yes	Opacity—White Lucency—Black	Fractures Dislocations Foreign bodies Bone infection Bone tumors
Computed Tomography (CT scan)	X-rays	2-D Slices	Yes X 100	Attenuation— High Low	3D-Reconstruction (Pelvic fracture, Pilon fracture, Tibial Plateau fracture, Acetabular fracture) Stress fracture Spine fracture Osteoid osteoma Bone tumors Tarsal coalition CT-guided biopsy
Ultrasound (US scan)	Sound Waves	Sector	No	Echogenicity— Hypo/Hyper +/- Acoustic Enhancement/ Shadow	DDH Abscess—Psoas Fracture healing—Ilizarov Cysts—Baker's cyst Bursitis Muscle tear Tendon rupture Joint effusion Foreign bodies DVT (Doppler US)
Magnetic Resonance Imaging - (MRI scan)	Rf-Radio-Frequency Pulse on tissues in Magnetic Field	Any slice Any plane	No	Signal Intensity Hypo (Low-black) Hyper (High-white) T1 and T2 Images T1-fat is bright T2-water is bright	Tumours staging Infection Spine-pathology Soft tissue injury-(Rotator cuff tear, Labral lesion, Menisci /cruciates) DDH Osteonecrosis Tarsal coalition
Bone Scan - Radionuclide Scan	Radioactive Tracer Technetium 99 Gallium 67 Indium 111	2-D A/P View of entire skeleton	Yes	Increased Activity— Hot scan or Cold scan	Bony Metastasis Stress fracture Bone infection Tumors AVN RSD
Bone Densitometry	X-rays	Density Pattern	Yes	Bone mineral content and density	Osteopenia Osteoporosis

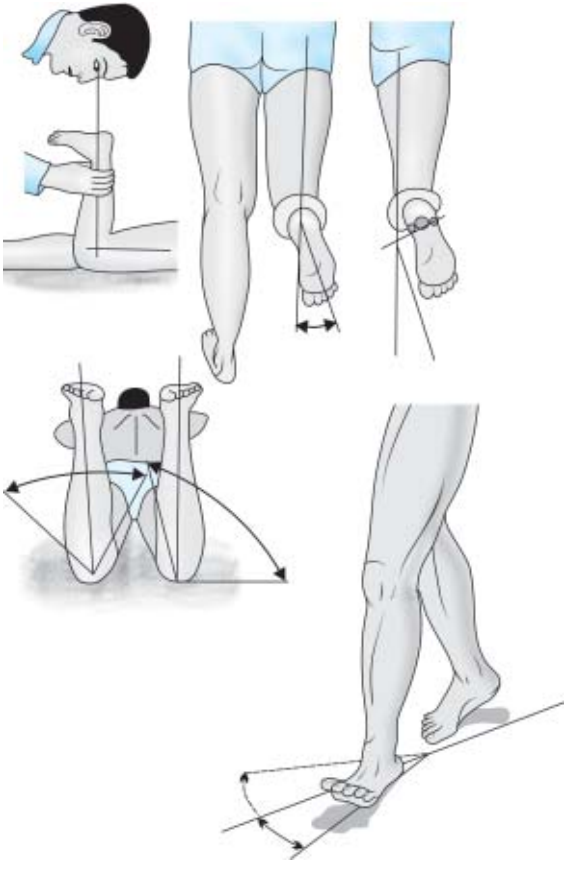


Fig. 14.1: Staheli's tests

1. *Foot progression angle (FPA)*: It is the angle between the longitudinal axis of foot and direction of gait progression. The average FPA is +4.2° with a normal range of -8° to +8°. It gives a measure of degree of toeing in or out due to contributions from foot, tibia and femur.
2. *Medial and lateral rotation of hip*: This is best assessed in prone position with knees flexed to 90°. Patient with excessive femoral anteversion will have more internal rotation movement with corresponding limitation of external rotation. Internal rotation of hip greater than 70° indicates excessive femoral anteversion. The version is estimated by the degree of hip rotation required to bring head and neck axis to the horizontal and is represented by the angle between the leg and vertical axis. Clinically the head and neck axis is made horizontal to the couch by rotating the leg and holding it when the trochanter is most prominent. The normal anteversion is about 40° at birth and 15° in adults.
3. *Thigh foot angle*: This determines the tibial torsion and is between the longitudinal axis of the thigh and foot when observed from above with patient in prone position and knee flexed to 90° (Normal 0 to 30°). Transmalleolar angle can also be used for tibial torsion; with patient in same position the angle between axis of thigh and line perpendicular to transmalleolar axis (line connecting mid point of medial and lateral malleoli) measures the tibial torsion. This can be used when there is a foot deformity **(Fig. 14.2)**.
4. The foot deformity is assessed by noting the position of forefoot in relationship to hind foot by the heel bisector line as described in the foot chapter.

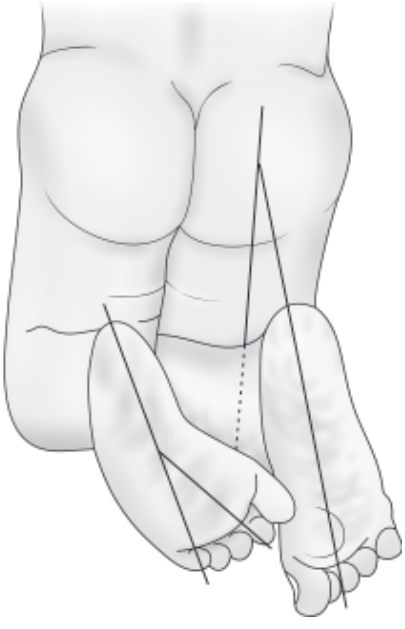


Fig. 14.2: Thigh foot angle and hindfoot forefoot angle

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